

Control of Communicable Diseases in Man

Ninth Edition

1960

**an Official Report of the
Indian Public Health Association**

CFTRI-MYSORE

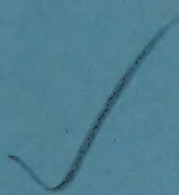
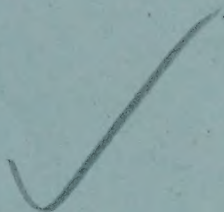


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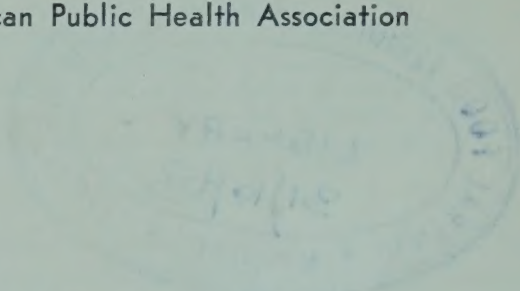
Control of Communicable Diseases in Man

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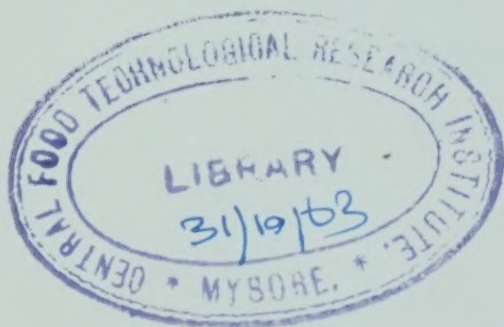
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This Report has been Approved in Principle by
The United States Public Health Service
and The Surgeons General of the United States Army,
Navy, and Air Force,
and has been endorsed by the
Association of State and Territorial Health Officers



The substance of this report has been accepted by the Ministry of Health for England and Wales and the Department of Health for Scotland with certain reservations to meet international commitments and agreements and differences in legislative and administrative practice in Great Britain.

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CONTROL OF COMMUNICABLE DISEASES IN MAN

Preface to the Ninth Edition

THE first edition of this official report of the American Public Health Association was prepared by a Committee of the Health Officers Section under the chairmanship of Haven Emerson, M.D., and published October 12, 1917. In 1935 responsibility for the text passed to the Committee on Research and Standards, which in 1957 became the Committee on Evaluation and Standards. Revisions by the Subcommittee on Communicable Disease Control of those two committees have appeared at 5-year intervals. The present ninth edition was prepared by the subcommittee with the following membership:

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Committee members and consultants took part in exchange of documents and in the meetings of October, 1958, in St. Louis, Mo., and in Atlantic City, N. J., in 1959, whereby common agreement was reached as to the facts and opinions to be presented and the form of their expression. This is the fourth edition in which the Ministry of Health for England and Wales and the Department of Health for Scotland have collaborated, and the third in which the World Health Organization has participated.

Purposes of the Manual—The aim is to provide an informative text for ready reference by public health workers of official and

voluntary health agencies, to include physicians, dentists, veterinarians, sanitary engineers, public health nurses, social workers, health educators, and sanitarians; and for physicians, dentists, and veterinarians in private practice having a concern with the control of communicable disease. The booklet is also designed for military physicians and others serving with the armed forces at home and abroad, and for health workers stationed in foreign countries. School administrators, medical students, and students of public health are others finding use for the material presented. The stated aim determines the format of the manual and its pocket size. The booklet does not substitute for standard textbooks. To meet the defined uses, the content is broadly representative of global infectious disease. While primarily intended for workers in the Americas and therefore largely expressive of American practices and ideas, a wider usefulness of the publication is seen in its translation into French, German, Spanish, Portuguese, Serbo-Croatian, Japanese, Chinese, Thai, and Finnish.

A second general purpose is to serve public health administrators as a guide and as a source of materials in preparing regulations and legal requirements for the control of the communicable diseases, in development of programs for health education of the public, and in the administrative acts of official health agencies toward management of communicable disease. The needs of field workers have had special attention through inclusion of much information applicable to field operations.

The intent is to present factual knowledge in brief fashion and to advance opinion consistent with those facts as a basis for intelligent management of communicable disease, unhampered by local usage and not restricted to prevailing practices. Recommendations toward standard administrative or technical procedure are avoided. The emphasis is on principle because local conditions and interrelated problems commonly require variation in practices from state to state within the United States, and between countries. Because differences in procedure are also due to incomplete knowledge of recent advances, and of practices proved successful under other and similar conditions, attempt is made to keep facts and opinions reasonably current by revision of the manual every 5 years.

Scope and Content—The report originally was designed as an aid in formulating standard regulations for administrative control of communicable diseases for which notification was required by state

and municipal health authorities of the United States. Because of overseas military activities and increasing international health activities by American workers, succeeding editions gave a more comprehensive coverage of world infections. The present list is by no means complete, but most conditions of public health importance are included, with a goodly representation of the less common and less significant diseases which so often present puzzling problems in administrative action.

As in earlier editions, the terms used are first defined. Each disease is briefly identified with regard to clinical nature, laboratory diagnosis, and differentiation of allied or related conditions. Occurrence, infectious agent, reservoir and source of infection, mode of transmission, incubation period, period of communicability, and susceptibility and resistance are next presented. Methods of control are described under the following 4 headings:

- A. *Preventive Measures:* Applicable generally to individuals and groups when and where the particular disease may occur in sporadic, endemic, or epidemic form, and whether or not the disease is an active threat at the moment, e.g., vaccination against smallpox, chlorination of water supplies, pasteurization of milk, control of rodents and arthropods, animal control, and immunization.
- B. *Control of Patient, Contacts, and the Immediate Environment:* Those measures designed to prevent infectious matter present in the person and environment of the infected individual from being conveyed to other persons, arthropods or animals in a way to spread the disease, and to keep contacts under surveillance during the assumed period of incubation of the disease and carriers under control until they are found to be free of the infectious agent of the disease in question. Specific treatment is included under this heading, as a means to limit period of communicability.
- C. *Epidemic Measures:* Those procedures of emergency character designed to limit the spread of a communicable disease which has developed widely in a group or community or within an area, state, or nation, such measures being unnecessary or not justified when the disease occurs sporadically or separated by considerable intervals in time, and only among widely separated individuals.

D. *International Measures:* Such controls of international travelers, immigrants, goods, animals and animal products, and their means of transport, as may arise from provisions of International Sanitary Regulations, conventions, intergovernmental agreements or national laws; also, any controls which may protect populations of one country against the known risk of infection from another country where a disease may be present in endemic or epidemic form. Immunization of persons and animals, deratting, control of arthropods, the quarantine and surveillance of travelers, and international exchange of epidemiological information are included.

Reporting of Communicable Disease—Notification to the local health authority that a case of communicable disease exists within the particular jurisdiction is the first step toward control. Administrative practice as to what diseases are to be reported and how they should be reported varies greatly from one region to another because of different conditions and different frequencies of disease. This manual presents a basic scheme of reporting directed toward practical working procedure rather than ideal practice. The purpose is encouragement of uniformity in morbidity reporting, to permit comparison of data within a country and between nations.

A system of reporting functions at 4 levels. The first is collection of the basic data in the local community where disease occurs. The data are next assembled at district, state, or provincial level. The third step is the collection of total information under national auspices. Finally, for certain prescribed diseases report is made by the national health authority to the World Health Organization.

Consideration is here limited to the first stage of a reporting system, the collection of the basic data at local level; first because that is the fundamental part of any scheme, and second because this manual is primarily for local health workers. The basic data sought at local level are of two kinds (Definition 33, Report of a Disease, p. 15).

1. **Report of Cases.** Each local health authority, in conformity with regulations of higher authority, will determine what diseases are to be reported as a routine and regular procedure, who is responsible for reporting, the nature of the report required and the manner in which reports are forwarded to the next superior jurisdiction.

Physicians are required to report all notifiable illnesses which come to their attention; in addition, the statutes or regulations of many localities require reporting by hospital, health department, or other person having knowledge of a case of a reportable disease.

Case Report of a communicable disease provides minimal identifying data of name, address, diagnosis, age, sex, and date of report for each patient and in some instances for suspects; dates of onset and of diagnosis are useful.

Collective Report is the assembled number of cases by diagnosis occurring within a prescribed time and without individual identifying data, e.g., 20 cases of malaria, week ending October 6.

2. Report of Epidemics. In addition to requirement of individual case report, any unusual or group expression of illness which may be of public concern (Definition 10, Epidemic, p. 12) is desirably reported to the local health authority by the most expeditious means, whether well known and subject to routine report, indefinite or unknown, or absent from the list of diseases officially reportable in the particular locality. Pertinent data of the Epidemic Report include number of cases, within what time, approximate population involved, and apparent mode of spread. The report is forwarded to next superior jurisdiction by telephone or telegraph.

Aside from the recommended requirement on Report of Epidemics, just stated, the communicable diseases listed in this manual are distributed among the following 5 classes, according to desirability and practical benefit presumably to be derived from reporting. These classes are referred to by number throughout the text, under Section 9B1 of each disease. The purpose is to provide a basis by which each health jurisdiction may determine its list of regularly reportable diseases.

Class 1. *Case Report Universally Required by International Sanitary Regulations*

This class is limited to the 6 internationally quarantinable diseases, which are cholera, plague, louse-borne relapsing fever, smallpox, louse-borne typhus fever, and yellow fever.

Obligatory case report to local health authority by telephone, telegraph, or other rapid means. The local health authority forwards the report to next superior jurisdiction by similar method if it is the first recognized case in the local area, or the first case outside those limits of the local area already affected; otherwise weekly by mail, or telegraphically in unusual situations.

Class 2. *Case Report Regularly Required Wherever the Disease Occurs*

Two subclasses are recognized, based on relative urgency for investigation of contacts and source of infection, or for starting control measures.

- A. Case report by telephone, telegraph, or other rapid means; forwarded to next superior jurisdiction weekly by mail, except that first recognized case in territory or first case outside limits of local area already affected is reported by telegraph; examples, typhoid fever, diphtheria.
- B. Case report by most practicable means; forwarded to next superior jurisdiction as a collective report, weekly by mail; examples, brucellosis, leprosy.

Class 3. *Selectively Reportable in Recognized Endemic Areas*

In many states and countries diseases of this class are not reportable. Reporting may be prescribed in particular regions, states, or countries by

reason of undue frequency or severity. Where applicable to the United States, USA is specified after "endemic areas." Three subclasses are recognized; A and B are primarily useful under conditions of established endemicity as a means toward prompt control measures and to judge effectiveness of control programs. The main purpose of C is to stimulate control measures or to acquire essential epidemiological data.

- A. Case report by telephone, telegraph, or other rapid means in specified areas where the disease ranks in importance with Class 2A; not reportable in many countries; examples, tularemia, scrub typhus.
- B. Case report by most practicable means; forwarded to next superior jurisdiction as a collective report by mail weekly or monthly; not reportable in many countries; examples, bartonellosis, coccidioidomycosis.
- C. Collective report weekly by mail to local health authority; forwarded to next superior jurisdiction by mail weekly, monthly, quarterly, or sometimes annually; examples, clonorchiasis, phlebotomus fever.

Class 4. Obligatory Report of Epidemics; No Case Report Required

Prompt report of outbreaks of particular public health importance by telephone, telegraph, or other rapid means; forwarded to next superior jurisdiction by telephone or telegraph. Pertinent data include number of cases, within what time, approximate population involved, and apparent mode of spread; examples, food poisoning, influenza.

Class 5. Official Report Not Ordinarily Justifiable

Diseases of this class are of two general kinds: those typically sporadic and uncommon, often not directly transmissible from man to man (blastomycosis); or of such epidemiological nature as to offer no practical measures for control (common cold).

Diseases are often made reportable although the information gathered is put to no practical use. This frequently has the result that the general level of reporting deteriorates, even for diseases of much importance. Better case reporting is usually to be had by restricting official report to those diseases for which control services are provided, or potential control procedures are under evaluation, or epidemiological information is needed for a definite purpose.

ACKNOWLEDGMENTS. Grateful acknowledgment is made for expert opinion and critical comment from many physicians and others, both within and without the Association. Diseases common to animals and man have had the benefit of veterinary opinion. Colleagues in other countries have been consulted on all diseases absent or not commonly present in the United States, and in many instances about communicable diseases of broader distribution where knowledge and opinion are incompletely defined. Their experience in research and control has contributed greatly to the accuracy and usefulness of this report.

TABLE OF CONTENTS

	<i>Page</i>
Preface	1
Definitions	11
Actinomycosis	19
African Tick-Borne Fever, Rickettsial (Boutonneuse Fever, South African Tick-Bite Fever, African Tick Typhus, Kenya Dog Fever)	20
Amebiasis (Amebic Dysentery)	21
Ancylostomiasis (Uncinariasis, Hookworm Disease)	23
Anthrax (Malignant Pustule, Charbon)	25
Arthropod-Borne Viral Infections—A Summary	27
Arthropod-Borne Viral Encephalitides	30
Arthropod-Borne Viral Fevers	33
Arthropod-Borne Viral Hemorrhagic Fevers	34
Ascariasis	38
Aseptic Meningitis Syndrome (Nonbacterial Meningitis, Viral Meningitis, Serous Meningitis)	39
Bartonellosis (Oroya Fever, Verruga Peruana)	41
Blastomycosis, North American	42
Blastomycosis, South American (Paracoccidioidal Granuloma)	43
✓ Botulism	44
Brucellosis (Undulant Fever, Malta Fever, Mediterranean Fever)	45
Candidiasis (Moniliasis, Thrush)	47
Cat Scratch Fever (Benign Inoculation Lymphoreticulosis)	48
Chancroid (Ulcus Molle, Chancre Mou, Soft Chancre, Soft Sore)	50
✓ Chickenpox (Varicella)	51
✓ Cholera	52
Chromoblastomycosis (Chromomycosis, Dermatitis Verrucosa)	54
Clonorchiasis (Chinese Liver Fluke Disease)	55
Coccidioidomycosis (Coccidioidal Granuloma, Valley Fever)	56
Colorado Tick Fever, Viral	58
Conjunctivitis, Acute Bacterial (Sore Eyes, Pink Eye)	59
Cryptococcosis (Torulosis, European Blastomycosis)	61
Dengue (Breakbone Fever)	62
✓ Diarrhea of the Newborn, Epidemic	63
✓ Diphtheria	66
Diphyllobothriasis (Broad or Fish Tapeworm Infection)	69
Dracontiasis (Guinea Worm Disease)	70
✓ Dysentery, Bacillary (Shigellosis)	71
Echinococcosis (Hydatidosis, Echinococciasis)	73
Enterobiasis (Pinworm or Seatworm Disease, Oxyuriasis)	75
Fasciolopsiasis	76
✓ Filariasis	78
Food Poisoning, Staphylococcal	80

	<i>Page</i>
Glanders	81
Gonococcal Infection	83
A. Gonococcal Urethritis (Gonorrhea)	84
B. Gonococcal Vulvovaginitis of Children	85
C. Gonococcal Ophthalmia Neonatorum (Gonorrheal Ophthalmia, Acute Conjunctivitis of the Newborn)	86
Granuloma Inguinale (Donovaniasis, Ulcerating Granuloma, Granuloma Pudendi Tropicum, Granuloma Contagiosa, Chronic Venereal Sore, Granuloma Venereum)	88
Hepatitis, Infectious (Epidemic Hepatitis, Epidemic Jaundice, Catarrhal Jaundice)	89
Hepatitis, Serum (Homologous Serum Jaundice)	90
Herpangina	91
Histoplasmosis	92
Influenza	94
Keratoconjunctivitis, Epidemic (Infection, Punctate Keratitis)	97
Larva Migrans, Visceral	98
Leishmaniasis, Cutaneous (In Old World: Aleppo, Bagdad or Delili Boil, Oriental Sore; in New World: Erazma, Uta, Chichero or Barro Ulcer)	100
Leishmaniasis, Visceral (Kala Azar)	102
Leprosy	103
Leptospirosis (Weil's Disease, Canicola Fever, Swinherd Disease, Mial Fever, Hemorrhagic Jaundice, Fort Bragg Fever, Rice Field Fever, and Cane Field Fever).....	105
Loiasis	107
Lymphocytic Choriomeningitis	108
Lymphogranuloma Venereum (Lymphogranuloma Inguinale, Climatic Bubo, Lymphopathia Venereum).....	109
Malaria	110
Measles (Rubeola, Morbilli)	113
Meningitis, Meningococcal (Cerebrospinal Fever)	115
Mononucleosis, Infectious (Glandular Fever, Monocytic Angina)	117
Mumps (Infectious Parotitis)	118
Nocardiosis	120
Onchocerciasis	120
Paragonimiasis (Pulmonary Distomiasis, Endemic Hematuria, Oriental Lung Fluke Disease)	122
Paratyphoid Fever	123
Pediculosis (Lousiness)	123
Phlebotomus Fever (Pappataci Fever, Sandfly Fever, Three-Day Fever)	124
Pinta (Mal del Pinto, Carate, Tinta, Lata, Fomequina, Anil)	125
Plague	127

	<i>Page</i>
Pleurodynia (Epidemic Pleurodynia, Bornholm Disease, Epidemic Myalgia, ✓ Devil's Grippe)	131
✓ Pneumonia	132
A. Pneumococcal—Acute Lobar Pneumonia	132
B. Bacterial Pneumonia, other than Pneumococcal	134
C. Primary Atypical Pneumonia (Virus Pneumonia)	135
Poliomyelitis (Infantile Paralysis)	137
Psittacosis (Ornithosis)	140
Q Fever	141
Rabies (Hydrophobia)	143
Rat-Bite Fever	145
A. Streptobacillus Moniliformis Infection (Haverhill Fever)	145
B. Spirillum Minus Infection (Sodoku)	146
Relapsing Fever, Louse-Borne	147
Relapsing Fever, Tick-Borne	148
Rheumatic Fever (Acute Articular Rheumatism)	150
Rickettsialpox	151
Rift Valley Fever	152
Ringworm (Favus, Jockey-Itch, Tinea, Athlete's Foot, The Dermatomy- coses, The Dermatophytoses)	154
A. Ringworm of Scalp (Tinea Capitis)	154
B. Ringworm of Body (Tinea Corporis)	156
C. Ringworm of Foot (Tinea Pedis)	157
D. Ringworm of Nails (Tinea Unguium)	158
Rocky Mountain Spotted Fever	159
Rubella (German Measles)	160
✓ Salmonellosis	162
Scabies	164
Schistosomiasis (Bilharziasis)	165
Scrub Typhus (Tsutsugamushi Disease, Mite-Borne Typhus Fever)	167
✓ Smallpox (Variola)	169
Sporotrichosis	172
Staphylococcal Infection	173
A. Impetigo Contagiosa	173
B. General Staphylococcal Infections Often Hospital-Acquired	175
Streptococcal Infection, Hemolytic	177
A. Scarlet Fever and Streptococcal Sore Throat (Streptococcal Tonsillitis, Streptococcal Pharyngitis)	177
B. Erysipelas	180
C. Streptococcal Puerperal Fever (Puerperal Septicemia)	182
Strongyloidiasis	184
Syphilis	185
A. Venereal Syphilis	185
B. Nonvenereal Syphilis (Bejel, Dichuchwa, Njovera, Sibbens, Radesyke)	188
Taeniasis and Cysticercosis (Beef or Pork Tapeworm Infection)	189
✓ Tetanus	190

	<i>Page</i>
Toxoplasmosis	192
Trachoma	194
Trichinosis (Trichiniasis, Trichinellosis)	195
Trichomoniasis	197
Trichuriasis (Trichocephaliasis, Whipworm Disease)	198
Trypanosomiasis, African (African Sleeping Sickness)	199
Trypanosomiasis, American (Chagas Disease)	201
Tuberculosis	203
Tularemia	207
✓ Typhoid Fever (Enteric Fever, Typhus Abdominalis)	209
Typhus Fever, Endemic Flea-Borne (Murine Typhus)	211
Typhus Fever, Epidemic Louse-Borne (Typhus Exanthematicus, Classical Typhus Fever)	212
Upper Respiratory Disease, Acute (Adenovirus and Unknown Etiology) ..	214
A. Pharyngoconjunctival Fever (Nonbacterial Pharyngitis, Nonbacterial Exudative Tonsillitis and Pharyngitis, Catarrhal Conjunctivitis)	215
B. Acute Respiratory Disease (ARD) of Military Recruits (Catarrhal Fever, Febrile Catarrh)	216
C. Acute Febrile Respiratory Diseases of Unknown Etiology	217
D. Common Cold	218
✓ Whooping Cough (Pertussis)	219
Yaws (Frambesia Tropica, Pian, Bouba, Parangi)	221
Yellow Fever	223
Index	227

DEFINITIONS

(Technical meaning of terms used in the text)

1. **Carrier**—A carrier is a person who harbors a specific infectious agent in the absence of discernible clinical disease and serves as a potential reservoir or source of infection for man. The carrier state may occur with infections inapparent throughout their course (commonly known as healthy carriers), and also as a feature of incubation period, convalescence, and postconvalescence of infections having a recognized clinical stage (commonly known as incubatory and convalescent carriers). Under either circumstance the carrier state may be short or long (temporary or chronic carriers). The same applies to animal carriers.

2. **Cleaning**—The removal from surfaces by scrubbing and washing, as with hot water, soap, and washing soda (Na_2CO_3) or other detergent, of infectious agents and of organic matter on which and in which infectious agents may find favorable conditions for prolonging life and virulence.

3. **Communicable disease**—An illness due to a specific infectious agent or its toxic products, arising through transmission of that agent or its products from reservoir to susceptible host, either directly as from an infected person or animal, or indirectly through the agency of an intermediate plant or animal host, a vector, or the inanimate environment. This report includes also an infestation by an ectoparasite, pediculosis (see 22, Infestation).

4. **Communicable period**—The time or times during which the infectious agent may be transferred directly or indirectly from an infected person to another person, or from an infected animal to man.

In diseases such as diphtheria and scarlet fever, in which mucous membranes are involved from the first entry of the pathogen, the period of communicability is from the date of first exposure to a source of infection, until the infecting microorganism is no longer disseminated from the involved mucous membranes; i.e., from before the prodromata until the termination of a carrier state, if such develops.

In diseases such as tuberculosis, syphilis, and gonorrhea, the communicable state may be at any time over a long and sometimes intermittent period when unhealed lesions of the disease permit the discharge of infectious agents from the surface of the skin or through any of the body orifices. In certain diseases communicability does not occur during the early incubation period or after full recovery; e.g., measles and chickenpox.

In diseases transmitted by arthropods, such as malaria and yellow fever, the periods of communicability are those during which the infectious agent occurs in the blood or other tissues of the infected person in infective form and in sufficient numbers for vector infection. A period of communicability is also to be distinguished for the arthropod vector, namely that time during which the agent is present in the tissues of the arthropod in such form as to be capable of transmitting infection.

5. **Contact**—A contact is any person or animal known to have been in such association with an infected person or animal as to have had the opportunity of acquiring the infection. Exposure may be immediate or intimate, involving touching or close approximation between infected individual and the contact, or remote or casual with no physical contact. Contacts thus are variously characterized as immediate, intimate, remote, or casual. Familial contacts are those living within the household of an infected person, as distinguished from those where exposure was outside the household group, in the course of work, school, or play.

6. *Contamination*—The presence of an infectious agent on a body surface, or on or in an inanimate article or substance.

7. *Disinfection*—Killing of infectious agents outside the body by chemical or physical means directly applied.

Concurrent disinfection is the application of disinfection as soon as possible after the discharge of infectious material from the body of an infected person, or after the soiling of articles with such infectious discharges, all personal contact with such discharges or articles being prevented prior to such disinfection.

Terminal disinfection indicates the process of rendering the personal clothing and immediate physical environment of the patient free from the possibility of conveying the infection to others after the patient has been removed, or has ceased to be a source of infection, or after isolation practices have been discontinued.

8. *Disinfestation*—Any physical or chemical process serving to destroy undesired animal forms, especially arthropods or rodents, present upon the person, the clothing, or in the environment of an individual, or on domestic animals. (See 23, Insecticide and 36, Rodenticide infra.) This includes the processes commonly called "dclousing" as applied to infestation with *Pediculus humanus*, the body louse.

9. *Education in personal cleanliness*—This phrase includes the various means available to impress upon all members of the community, young and old, and especially when communicable disease is prevalent or during epidemics, by spoken and printed word, and by illustration and suggestion, the necessity of:

- a. Keeping the body clean by sufficiently frequent soap and water baths.
- b. Washing hands in soap and water immediately after voiding bowels or bladder and always before eating.
- c. Keeping hands and unclean articles, or articles that have been used for toilet purposes by others, away from the mouth, nose, eyes, ears, genitalia, and wounds.
- d. Avoiding the use of common or unclean eating, drinking, or toilet articles of any kind, such as cutlery and crockery, drinking cups, towels, handkerchiefs, combs, hairbrushes, and pipes.
- e. Avoiding exposure of persons to spray from the nose and mouth, as in coughing, sneezing, laughing, or talking.
- f. Washing hands thoroughly after handling the patient or his belongings, and wearing a protective overall apron while in the sickroom.

10. *Epidemic*—An epidemic or outbreak is here defined as the occurrence in a community or region of a group of illnesses of similar nature, clearly in excess of normal expectancy, and derived from a common or from a propagated source. The number of cases indicating presence of an epidemic will vary according to the infectious agent, size and type of population exposed, previous experience or lack of exposure to the disease, and time and place of occurrence; epidemicity is thus relative to usual frequency of the disease in the same area among the specified population at the same season of year. A single case of a communicable disease long absent from a population (as dengue fever in Florida) or first invasion by a disease not previously recognized in that area (as American trypanosomiasis in Arizona) is to be considered as a potential epidemic meeting the requirements in respect to reporting of epidemics.

11. *Fatality*—The number of deaths in relation to numbers of patients or cases in which these deaths occur, usually used as disease-specific and usually expressed as a ratio in terms of per cent. Thus, the fatality of diphtheria is 1/100.

12. **Fumigation**—Any process by which the killing of animal forms, especially arthropods and rodents, is accomplished by the employment of gaseous agents. (See 23, Insecticide and 36, Rodenticide.)

13. **Host**—A man or other living animal, including birds and arthropods, affording under natural conditions subsistence or lodgment to an infectious agent. Some protozoa and helminths pass successive stages in alternate hosts of different species. Hosts in which the parasite attains maturity or passes its sexual stage are primary or definitive hosts; those in which the parasite is in a larval or asexual state are secondary or intermediate hosts.

14. **Immune person**—An immune person is one who possesses specific protective antibodies or cellular immunity as a result of previous infection or immunization, or is so conditioned by such previous specific experience as to respond adequately with production of antibodies sufficient in either instance to protect from illness following exposure to the specific infectious agent of the disease. This applies also to animals. Immunity is relative; an ordinarily effective protection may be overwhelmed by an excessive dose of the infectious agent or an unusual portal of entry.

15. **Inapparent infection**—A person or animal has an inapparent infection (also known as latent or subclinical infection) when the infectious agent has so mild an effect that even though infection be present and is identifiable by laboratory means, it is undetected clinically.

16. **Incidence**—The number of cases of disease, of infection, or other event occurring during a prescribed time period, in relation to the unit of population in which they occur (a dynamic measurement); thus the incidence of tuberculosis expressed as a rate is the number of new cases reported per 100,000 population per year.

17. **Incubation period**—The time interval between the infection of a susceptible person or animal and the appearance of signs or symptoms of the disease in question.

18. **Infected person**—Infected persons include patients or sick persons, persons with inapparent infection, and carriers. The same expression holds for animals, including arthropods.

19. **Infection**—The entry and development or multiplication of an infectious agent in the body of man or animal. Infection is not synonymous with infectious disease; the result may be inapparent (see 15, Inapparent infection) or manifest (see 21, Infectious disease). The presence of living infectious agents on exterior surfaces of the body or upon articles of apparel or soiled articles, is not infection but contamination of such surfaces and articles. The term "infection" should not be used to describe conditions of inanimate matter such as soil, water, sewage, milk, or food; the term "contamination" applies (see 6, Contamination).

20. **Infectious agent**—A microorganism (bacterium, protozoon, helminth, spirochete, fungus, virus, or other) capable of producing infection, and under favorable circumstances of host and environment having the capacity to produce infectious disease.

21. **Infectious disease**—A disease of man or animal resulting from an infection.

22. **Infestation**—By infestation of persons and animals is meant the lodgment, development, and reproduction of arthropods on the surface of the body or in the clothing. Infested articles or premises are such as harbor or give shelter to animal forms, especially arthropods and rodents.

23. **Insecticide**—Any chemical substance used for the destruction of arthropods, whether applied as powder, liquid, atomized liquid, aerosol, or as a

paint-spray; residual action is usual. The term *larvicide* is generally used to designate insecticides applied specifically for destruction of immature stages of arthropods; *imagicide* and *adulticide*, to designate those applied to destroy mature and adult forms.

24. Isolation—The separation for the period of communicability of infected persons from other persons, in such places and under such conditions as will prevent the direct or indirect conveyance of the infectious agent from infected persons to other persons who are susceptible or who may spread the agent to others. This applies also to animals.

Strict isolation of the patient for the period of communicability is necessary in certain diseases, notably smallpox. However, isolation of the patient has but little effect in limiting the spread of many diseases, for instance poliomyelitis.

When used in connection with such diseases as the common cold, influenza, chickenpox, mumps, and the pneumonias, isolation is not to be understood, under ordinary circumstances, as a necessary or practicable procedure for official requirement or enforcement, but a modified practice to be instituted under the direction of the attending physician, and its duration to be generally, if not exclusively, at his direction.

Other than for rare exceptions and under special circumstances, the placarding of premises has no place in public health practice as a means to enforce isolation.

25. Morbidity—A general and variously used term expressing the number of sick persons or cases of disease in relation to the population in which they occur. Rates may be in terms of incidence (see Definition 16) or of prevalence (see Definition 30). Disease-specific incidence rates are common usage in expressing morbidity, sometimes further qualified for age, sex or other attribute, and usually representing cases per 100,000 population per year. Attack rate is an incidence rate often used for particular populations, observed for limited periods and under special circumstances, as in an epidemic; the secondary attack rate in communicable disease practice is the number of cases among familial or institutional contacts occurring within the accepted incubation period directly following exposure to a primary case. Case rate expresses the incidence of clinically recognized cases; infection rate the sum of infection and infectious disease. Admission rate is the usual term for morbidity in military populations, an incidence rate that includes both patients admitted to hospital and those confined to quarters.

26. Mortality The relation of deaths to the population in which they occur. The mortality rate (death rate) expresses the number of deaths in a unit of population within a prescribed time; for crude total rates usually the number of deaths per 1,000 population per year. Rates are commonly specific for disease and sometimes or additionally for age, sex or other attribute, again with the time one year and a population suited to the particular purpose, 1,000, 10,000, or commonly 100,000.

27. Molluscicide A chemical substance used for the destruction of snails, generally through ingestion.

28. Pathogenicity The capacity of microorganisms to cause disease or morbid symptoms. Virulence denotes the relative infectiousness of a microorganism or its ability to overcome the defenses of the host.

29. Patient or sick person—A person who is ill; here limited to a person suffering from a recognizable attack of a communicable disease.

30. Prevalence—The number of cases of disease, of infected persons, or of persons with some other attribute, present at a particular time and in relation to the size of the population from which drawn (a static measurement); thus the prevalence of tuberculosis is commonly expressed as the number of active

cases (all forms, old and new) existing at a designated time per 100,000 persons.

31. **Quarantine**—(1) Complete quarantine is the limitation of freedom of movement of such well persons or domestic animals as have been exposed to a communicable disease, for a period of time equal to the longest usual incubation period of the disease, in such manner as to prevent effective contact with those not so exposed.

(2) Modified quarantine is a selective, partial limitation of freedom of movement of persons or domestic animals, commonly on the basis of known or presumed differences in susceptibility, but sometimes because of danger of disease transmission. It may be designed to meet particular situations; examples are exclusion of children from school or exemption of immune persons from provisions required of susceptible persons, such as contacts acting as food handlers, or restriction of military populations to the post or to quarters.

(3) Surveillance is the practice of close supervision of contacts for purposes of prompt recognition of infection or illness but without restricting their movements.

(4) Segregation is the separation for special consideration, control, or observation of some part of a group of persons or of domestic animals from the others, to facilitate the control of a communicable disease. Removal of susceptible children to homes of immune persons, or the establishment of a sanitary boundary to protect uninfected from infected portions of a population are examples.

32. **Repellent**—A chemical applied to the skin or clothing or other places to discourage arthropods from lighting on and attacking an individual.

33. **Report of a disease**—Official report is notification of the occurrence of a communicable or other disease in man or animals; to the local health authority for diseases of man, to the livestock, sanitary or agriculture authority for disease of animals, and in some instances, disease of both animal and man to the health authority. Report should include suspect cases of diseases of particular public health importance, ordinarily those requiring epidemiologic investigation or initiation of special control measures.

When a person is infected in one health jurisdiction and the case is reported from another, the authority receiving the report should notify the other jurisdiction involved, especially if the disease is such as to require examination of contacts for infection, and of food or water supplies as vehicles.

In addition to routine report of specified diseases, as determined by the needs of the particular health jurisdiction, special notification of all epidemics or outbreaks of disease, including those not in the list declared reportable, is regularly to be required (see 10, Epidemic).

34. **Reservoir of infection**—Reservoirs of infection are man, animals, plants, soil, or inanimate organic matter, in which an infectious agent lives and multiplies and depends primarily for survival, reproducing itself in such manner that it can be transmitted to a susceptible host. Man himself is the most frequent reservoir of infectious agents pathogenic for man.

35. **Resistance**—The sum total of body mechanisms which interpose barriers to the progress of invasion of infectious agents.

a. **Immunity**—Immunity is here defined as that resistance usually associated with possession of antibodies having a specific action on the micro-organism concerned with a particular infectious disease. Passive immunity is attained either naturally by maternal transfer, or artificially by inoculation of specific protective antibodies (convalescent or immune serum, or gamma globulin) and is of brief duration. Active immunity is attained either naturally by infection, with or without

clinical manifestations, or artificially by inoculation of fractions or products of the infectious agent, or of the agent itself in killed, modified, or variant form.

- b. *Inherent insusceptibility*—Inherent insusceptibility is an ability to resist disease independently of antibodies or specifically developed tissue response; it commonly rests in anatomic or physiologic characteristics of the host; it may be genetic or acquired; permanent or temporary; autarcesis.

36. *Rodenticide*—A chemical substance used for the destruction of rodents, generally through ingestion. (Compare 12, Fumigation.)

37. *Source of infection*—The thing, person, object, or substance from which an infectious agent passes immediately to a host. Transfer is often direct from reservoir to host in which case the reservoir is also the source of infection (measles). The source may be at any point in the chain of transmission, as a vehicle, vector, intermediate animal host, or contaminated article; thus, contaminated water (typhoid), an infective mosquito (yellow fever), beef (tape worm infection), or a toy (diphtheria). In each instance cited the reservoir is an infected person. (Compare 34, Reservoir.)

38. *Susceptible*—A person or animal presumably not possessing resistance against a particular pathogenic agent and for that reason liable to contract a disease if exposed to such agent.

39. *Suspect*—A person whose medical history and symptoms suggest that he may have or be developing some communicable disease.

40. *Transmission of infection*—Modes of transmission are the mechanisms by which an infectious agent is transported from reservoir to susceptible human host. They are:

a. *Contact*:

- (1) *Direct contact*: Actual touching of the infected person or animal or other reservoir of infection, as in kissing, sexual intercourse, or other contiguous personal association.
- (2) *Indirect contact*: Touching of contaminated objects such as toys, handkerchiefs, soiled clothing, bedding, surgical instruments and dressings, with subsequent hand to mouth transfer of infective material; less commonly, transfer to abraded or intact skin or mucous membrane.
- (3) *Droplet spread*: The projection onto the conjunctivae and the face or into the nose or mouth of the spray emanating from an infected person during sneezing, coughing, singing or talking. Such droplets usually travel no more than 3 feet from the source. Transmission by droplet infection is considered a form of contact infection, since it involves reasonably close association between two or more persons.

b. *Vehicle*: Water, food, milk, biological products to include serum and plasma, or any substance serving as an intermediate means by which an infectious agent is transported from a reservoir and introduced into a susceptible host through ingestion, through inoculation, or by deposit on skin or mucous membrane.

c. *Vector*: Arthropods or other invertebrates which transmit infection by inoculation into or through the skin or mucous membrane by biting or by deposit of infective materials on the skin or on food or other objects. The vector may be infected itself (in this state termed *infective*) or may act only as a passive or mechanical carrier of the agent.

d. Air-Borne:

- (1) Droplet nuclei: The inhalation of the small residues which result from evaporation of droplets (see a(3) above) and remain suspended in air of enclosed spaces for relatively long periods of time. Droplet nuclei also may be created purposely by a variety of atomizing devices, or accidentally in the course of many laboratory procedures.
- (2) Dust: The inhalation or settling on body surfaces of coarser particles which may arise from contaminated floors, clothes, bedding or other articles, or from soil, and ordinarily remain suspended in the air for relatively short periods of time.

41. **Zoonosis**—An infection or an infectious disease of animals transmissible under natural conditions to man.

ACTINOMYCOSIS

1. **Identification**—A chronic suppurative or granulomatous process, most frequently localized in jaw, thorax, or abdomen, rarely limited to skin and subcutaneous tissues; characterized by firm induration, the granulomata spreading slowly to contiguous tissues and focally breaking down to form multiple draining sinuses which penetrate to the surface. Discharges from sinus tracts contain "sulfur granules" which are colonies of the infectious agent, soft at first, becoming firm to hard, reaching a diameter of 1 mm or more, with peripheral "clubs" composed of eosinophilic material deposited by the host about the radially oriented hyphal tips of the fungus.

Diagnosis is confirmed by anaerobic culture of the fungus on an enriched medium.

2. **Occurrence**—An infrequent disease of man, occurring sporadically all over the world. All races, both sexes, and all age groups (highest frequency 15 to 35 years of age) may be affected; the ratio of males to females is approximately two to one. Occurs also in cattle, swine, horses and other animals. There is no evidence of spread from animals to man.
3. **Infectious agent**—*Actinomyces israelii* and *A. bovis*, gram-positive non-acid-fast, anaerobic fungi.
4. **Reservoir and source of infection**—The natural reservoir is man. In the normal oral cavity *Actinomyces* grows as a saprophyte in and around carious teeth, in dental plaques, and in tonsillar crypts, without apparent penetration or cellular response of adjacent tissues. In sample surveys *A. israelii* has been demonstrated in smears from granules in the crypts of 40% of extirpated tonsils and isolated in pure anaerobic culture from 10%. No external environmental reservoir such as straw or soil has been demonstrated. The source of clinical disease is endogenous. Presumably a similar endogenous reservoir occurs in cattle and is similarly related to bovine actinomycosis, but transmission from cattle to man has not been demonstrated.
5. **Mode of transmission**—Presumably the fungus passes by contact from man to man as a part of the normal oral flora. From the oral cavity the fungus may be swallowed, inhaled, or introduced into jaw tissues by injury or at the site of a neglected or irritating dental defect.
6. **Incubation period**—Unknown; probably many years after appearance in the normal oral flora, and weeks or months after precipitating trauma and actual penetration of tissues.
7. **Period of communicability**—Time and manner in which *A. israelii* becomes a part of the normal oral flora unknown and not pertinent. Origin and development of clinical actinomycosis is endogenous and not dependent upon exposure to a person or animal with the disease.
8. **Susceptibility and resistance**—Natural susceptibility is low. Immunity following attack has not been demonstrated.
9. **Methods of Control**—
 - A. **Preventive measures**: None. In slaughter houses parts of carcasses or organs containing lesions should be destroyed as unfit for food.
 - B. **Control of patient, contacts, and the immediate environment**:
 1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).

2. Isolation: None.
3. Concurrent disinfection: Of discharges and contaminated dressings.
4. Terminal disinfection: Thorough cleaning.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Not profitable.
8. Specific treatment: No spontaneous recovery. Prolonged administration of sulfonamides, penicillin, chlortetracycline, or chloramphenicol is usually effective; prognosis poor in meningitis.

C. *Epidemic measures*: Not applicable, a sporadic disease.

D. *International measures*: None.

AFRICAN TICK-BORNE FEVER, RICKETTSIAL

1. **Identification**—A mild to moderately severe illness, characterized by an initial lesion associated with tender enlarged lymph nodes, a febrile period of about 10 days and a generalized maculopapular erythematous rash which appears about the fourth or fifth day, usually involves palms and soles and persists 6 to 7 days; with antibiotic treatment, fever lasts no more than 2 days. The fatality is less than 3%, even without specific therapy. Synonyms: Boutonneuse Fever, South African Tick-bite Fever, African Tick Typhus, Kenya Dog Fever.

Laboratory diagnosis is by animal inoculation or through demonstrating antibodies by serologic test using a specific rickettsial antigen. The Weil-Felix test (reaction with Proteus OX 19 and OX 2) is frequently positive, in about equal degree for the 2 antigens, with the end titer usually lower than in other rickettsial diseases, especially OX 19.

2. **Occurrence**—In Mediterranean and Black Sea areas and widely throughout Africa but not in the Western Hemisphere. In more temperate areas the highest incidence is during warmer months when ticks are numerous; in tropical areas throughout the year. Outbreaks may occur when groups of susceptibles are brought into an endemic area.
3. **Infectious agent**—*Rickettsia conori*, a member of the spotted fever group of rickettsiae.
4. **Reservoir and source of infection**—Reservoir is a tick, or a wild or field rodent. Infective ticks are the immediate source of infection.
5. **Mode of transmission**—In the Mediterranean area, by bite of infected *Rhipicephalus sanguineus* which infests dogs and their habitations. In South Africa a number of ticks are infected in nature and presumed to be vectors, viz., *Haemaphysalis leachi*, *Amblyomma hebraeum*, *Rhipicephalus appendiculatus*, *Boophilus decoloratus* and *Hyalomma aegyptium*.
6. **Incubation period**—Usually 5 to 7 days.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Ticks remain infective for life, commonly as long as 18 months; transovarian infection occurs.
8. **Susceptibility and resistance**—Susceptibility appears to be general; one attack confers immunity.

9. Methods of control—**A. Preventive measures:**

1. Personal prophylaxis is by avoiding tick-infested areas when feasible; or by taking precautions to prevent tick-bite by use of tick repellents (dimethylphthalate, diethyltoluamide); or by prompt removal of attached ticks without crushing and without contaminating the hands.
2. Attempts to reduce numbers of ticks in nature is generally impractical but in areas where dog and *R. sanguineus* are important members of the cycle, local measures are indicated, including stray dog control.
3. Vaccines are not available commercially.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in many countries not a reportable disease. Class 3B (p. 5).
2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Pest control measures against ticks infesting households and dogs.
8. Specific treatment: One of the tetracycline antibiotics, or chloramphenicol orally until afebrile (about 2 days) and for 1 additional day.

C. Epidemic measures: Rarely required; general application of measures listed under 9A1 and 9A2, careful observation of group at risk with prompt antibiotic therapy for those becoming ill, and disinfection of selected terrain by insecticides (see Rocky Mountain Spotted Fever, 9A2, p. 160).

D. International measures: Effort to prevent introduction of infected ticks and their vertebrate hosts into areas believed free of the disease.

AMEBIASIS

1. Identification—Amebic infection has a wide range of clinical manifestations. The primary site is the colon; infection may be asymptomatic, or with mild symptoms that include abdominal discomfort and diarrhea alternating with constipation, or a chronic diarrhea with mucus and some blood, or an acute dysentery with profuse blood and mucus, usually with little pus. Amebiasis is only occasionally a direct cause of death. Infection may spread by the blood stream or by direct extension, producing amebic hepatitis, abscess of liver, lung or brain, or ulceration of skin. Synonym: Amebic Dysentery.

Definitive diagnosis is through identifying trophozoites or cysts of *Entamoeba histolytica* in the feces, or trophozoites in smears or sections from lesions.

Differential diagnosis includes shigellosis, appendicitis, ulcerative colitis, balantidial and giardial diarrhea (balantidiasis, giardiasis). Other intestinal protozoa also are associated with diarrhea in man. These infections may exist concomitantly with *E. histolytica*.

2. Occurrence—Infection with *E. histolytica* is world-wide, often to the extent of 50% or more of people in unsanitated areas, especially in

tropical countries and in mental institutions; low (1 to 5%) in well-sanitized cities; clinical amebic dysentery is prevalent in warm and hot countries and relatively infrequent in temperate regions although infection rates may be high and water-borne epidemics occur occasionally.

3. Infectious agent—*Entamoeba histolytica*.

4. Reservoir and source of infection—Reservoir is an infected person, usually a chronic or asymptomatic case. Acute infections are of little menace because of fragility of trophozoites. Source of infection is cysts from feces.

5. Mode of transmission—Fresh fecal contamination; contaminated vegetables, especially those commonly served raw, cold, and moist; contaminated water; flies; soiled hands of infected food handlers.

6. Incubation period—From 5 days in severe infections to several months in subacute and chronic cases; commonly from 3 to 4 weeks.

7. Period of communicability—During intestinal infection, which may continue for years.

8. Susceptibility and resistance—Susceptibility to infection is general; relatively few persons harboring the organism develop recognized symptoms; acute cases tend to become chronic. Immunity to reinfection has not been clearly established in man. No artificial immunity.

9. Methods of control—

A. Preventive measures:

1. Sanitary disposal of human feces.

2. Protection of public water supplies against fecal contamination, and boiling drinking water where necessary. Chlorination of water supplies as generally practiced is inadequate for destruction of cysts; sand filtration removes nearly all cysts, diatomaceous earth filters completely. Avoidance of cross-connections between public and private auxiliary water supplies and of back flow connections in plumbing systems. Small quantities of water, as in Lyster bags and canteens, are best protected by tablets of tetraglycine hydroperiodide (globaline).

3. Supervision of general cleanliness, personal health, and sanitary practices of persons preparing and serving food in public eating places, especially moist foods eaten raw. Routine examination of food handlers as a control measure is impractical.

4. Disinfectant dips for fruits and vegetables are as yet of unproved value.

5. Fly control and protection of foods against fly contamination by screening or other appropriate measure.

6. Instruction of convalescents and of the general public in personal hygiene, particularly as to sanitary disposal of feces, and hand washing after defecation and before preparing or eating food.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas (USA); in many states and countries not a reportable disease, Class 3C (p. 5).

2. Isolation: None. Exclusion of patient from preparation, processing and serving of food until treatment is completed.

3. Concurrent disinfection: Sanitary disposal of feces. Hand washing after defecation.

4. Terminal disinfection: Cleaning.

5. Quarantine: None.
 6. Immunization of contacts: Not applicable.
 7. Investigation of contacts and source of infection: Microscopic examination of feces of members of household, and of other suspected contacts, supplemented by search for direct contamination of water by human feces.
 8. Specific treatment: Acute amebic dysentery: Tetracyclines and emetine hydrochloride are the drugs of choice. Emetine hydrochloride will relieve symptoms but will usually not eliminate the infection. Both should be accompanied or followed by a standard amebicide, such as a hydroxyquinoline, carbarsone ®, or milibis ®, to eliminate the infection from the lumen of the intestine. These amebicides also serve in treatment of mild and asymptomatic infections.
- Amebic hepatitis and liver abscess: chloroquine, aralen ® or emetine hydrochloride. If abscess requires aspiration, precede by chloroquine or emetine to limit infection. Broad spectrum antibiotics should be administered to control possible bacterial infection.
- Repeated fecal examination at intervals up to 3 months is necessary to assure that amebae have been eliminated.

C. Epidemic measures: Any grouping of several cases from a single area or in an institution requires prompt epidemiologic investigation to determine source of infection and mode of transmission. If a common vehicle is indicated, such as water or food, take appropriate measures to correct the situation. Kitchen sanitation is particularly important where general sanitary conditions are poor. It is important to provide conveniently available toilets and hand-washing facilities in restaurants or other community eating establishments and to teach food handlers to use them. If the epidemiologic evidence points to person-to-person transmission, the emphasis is on personal cleanliness, sanitary disposal of feces, and fly control.

D. International measures: None.

ANCYLOSTOMIASIS

1. **Identification**—A chronic debilitating infection caused by hookworms. A variety of vague symptoms occur, varying greatly according to extent of the infection and nutrition of the patient. The blood-sucking activity of the worms along with malnutrition lead to hypochromic microcytic anemia, a major cause of disability. Infected children may be retarded in mental and physical development. Death is infrequent either in acute or chronic stages, and then usually in association with other infection. Synonyms: Uncinariasis, Hookworm Disease.
- Hookworm infection is confirmed by finding the eggs of worms in feces; species recognition is through microscopic examination of adult worms.
2. **Occurrence**—Widely endemic in those tropical and subtropical countries where disposal of feces is inadequate, and soil, moisture, and temperature favor development of infective larvae. *N. americanus* is the prevailing species throughout tropical West Africa and southeastern United States; and *A. duodenale* in Mediterranean countries, including the Nile Valley. Both forms occur in many parts of Asia, Central and South America, and the West Indies.
3. **Infectious agents**—*Necator americanus* and *Ancylostoma duodenale*. Third-stage larvae pass from the skin via lymphatics and blood stream

to the lungs, enter the alveoli, migrate up the trachea to the pharynx, are swallowed, and reach the small intestine where they attach to the intestinal wall, develop to maturity and produce eggs.

Infective larvae of cat and dog hookworm (*Ancylostoma braziliense* and *A. caninum*) cause a dermatitis in man called creeping eruption, an occasional disease of bathers, utility men, gardeners, children, and others in contact with contaminated sandy soil; in the United States, mainly in the southeast. The larvae are destroyed in the skin and do not otherwise affect man.

4. **Reservoir and source of infection**—Reservoir is an infected person discharging eggs in feces. The usual source of infection is soil contaminated with infective larvae.
5. **Mode of transmission**—Eggs in feces are deposited on the ground, hatch, larvae develop to the third stage (infective form) and penetrate the skin, usually the foot, in so doing characteristically producing a dermatitis (ground itch). Infection via the alimentary tract is also possible.
6. **Incubation period**—The first eggs appear in the feces about 6 weeks after infection. Symptoms may appear after a few weeks to many months or even years, depending on intensity of infection.
7. **Period of communicability**—Infected persons are potential spreaders of infection as long as they remain infected and continue to pollute soil. Under favorable conditions third stage infective larvae remain alive in soil for several weeks.
8. **Susceptibility and resistance**—Susceptibility is universal but the disease in the United States is less frequent and less severe among Negroes than whites. Some immunity develops with infection.
9. **Methods of control**—

A. Preventive measures:

1. Education as to dangers of soil pollution and methods of prevention.
2. Prevention of soil pollution by installation of sanitary disposal systems for human discharges, especially sanitary privies in rural areas, and education of the public in the use of such facilities.
3. Personal prophylaxis by cleanliness and the wearing of shoes.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas (USA); in most countries not a reportable disease. Class 3C (p. 5).
2. Isolation: None.
3. Concurrent disinfection: Sanitary disposal of feces to prevent contamination of soil and water.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Each patient and carrier is a potential or actual spreader of the disease. Examine all family contacts.
8. Specific treatment: Tetrachlorethylene or hexylresorcinol; toxic reactions are infrequent and therapy can be repeated if necessary. The duration of communicability is shortened.

C. Epidemic measures: Surveys for prevalence in highly endemic areas, public health education in sanitation of the environment and in personal hygiene, and provision of facilities for treatment.

D. International measures: None.

ANTHRAX

1. **Identification**—An acute bacterial infection of animals and man; in man, usually a disease of the skin. An initial papule and vesicle at the site of inoculation develop into a black eschar, commonly followed by hard edematous swelling of deeper and adjacent tissues. Pain associated with the lesion is unusual, and if present is related to edema. If untreated, the infection may extend progressively to regional lymph nodes and bloodstream with overwhelming septicemia and death. Inhalation and gastrointestinal anthrax are rare; initial symptoms are mild and non-specific, resembling common upper respiratory infections, but acute symptoms of respiratory distress and shock follow in from 3 to 5 days, with death commonly 7 to 25 hours thereafter; characteristic post-mortem findings are hemorrhagic mediastinitis often accompanied by hemorrhagic meningitis. Untreated cutaneous anthrax has a fatality of from 5 to 20%; with effective antibiotic therapy, there should be no deaths. The other two forms are highly fatal. Synonym: Malignant Pustule, Charbon.

Laboratory confirmation is by direct demonstration of bacilli in lesions or discharges; by culture, by inoculation of mice or guinea pigs with exudates from lesions, or with blood or other tissues, or with pure cultures; and by use of *Bacillus anthracis* bacteriophage. Fluorescent antibody technics can be used for visualization and identification of the organism in tissue sections.

2. **Occurrence**—Infrequent and sporadic in man; primarily an occupational hazard of veterinarians and agricultural workers handling infected animals, and of industrial workers processing hides, hair, and wool. Endemic in agricultural regions favorable to the organism, the requirements of which are suggestively strict, with formation of "anthrax districts." New areas may develop through introduction of contaminated feed or altered environmental conditions such as floods and drought.
3. **Infectious agent**—*Bacillus anthracis*. The ability to form spores permits the organism to survive environmental and disinfective measures destroying most other bacteria; the public health problems largely arise from this circumstance.
4. **Reservoir and source of infection**—The reservoir is any one of several species of animals: cattle, sheep, goats, horses, pigs, and others. The source of infection is the tissues of animals dying of the disease; or contaminated hair, wool, hides, and soil associated with the infected animal.
5. **Mode of transmission**—Infection of skin is by hair, wool, hides, contaminated shaving brushes, and other manufactured products; or by direct contact with infected tissues. Primary inhalation anthrax when it occurs, presumably results from inhalation of spores. Gastrointestinal anthrax results from ingestion of contaminated meat; milk is not involved. The disease spreads among omnivorous and carnivorous animals through contaminated meat, bone meal, or other feed products. Flies may serve as mechanical carriers. Vultures have spread the infection from one area to another. Accidental infections occur among laboratory workers.
6. **Incubation period**—Within 7 days, usually less than 4.
7. **Period of communicability**—Rarely if ever transmitted under natural conditions from man to man, although discharges from lesions contain anthrax bacilli. Contaminated articles and soil may remain infective for years.

8. **Susceptibility and resistance**—Uncertain. There is no evidence of inapparent infection among persons in frequent or continued contact with the infectious agent. Second attacks have been reported, but none are well documented.

9. **Methods of control**—

A. Preventive measures:

1. Animals suspected of anthrax should be promptly isolated and treated. Milk of infected animals rarely transmits anthrax; avoid human use but may be boiled and fed to swine or chickens.
2. Post-mortem examination of animals dying of suspected anthrax should be made by a veterinarian, and with care not to contaminate soil or environment with blood or infected tissues. Carcasses should be completely burned or buried deeply with quick lime.
3. Annual inoculation of animals in enzootic areas with an approved vaccine.
4. Control of effluents and trade wastes of rendering plants handling potentially infected animals, and of factories that manufacture products from hair, wool or hides likely to be contaminated.
5. Education in personal cleanliness and care of skin abrasions of employees handling potentially contaminated articles. Continuing medical supervision of employees, with prompt medical care of all suspicious skin lesions to exclude anthrax.
6. Dust control and proper ventilation in hazardous industries.
7. Thorough washing, disinfection or sterilization, when possible, of hair, wool or hides, and bone meal or other feed of animal origin, prior to processing.
8. Hides of animals infected with anthrax must not be sold, nor carcasses used as food.
9. A cell-free antigen is currently being evaluated as an immunizing agent in industrial situations; it appears effective in preventing cutaneous anthrax. When available, it is recommended for employees handling potentially contaminated industrial raw materials.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report obligatory in most states and countries, Class 2A (p. 5). Report also to appropriate livestock sanitary or agriculture authority.
2. Isolation: Until lesions are bacteriologically free of anthrax bacilli.
3. Concurrent disinfection: Of discharges from lesions and articles soiled therewith. Spores require steam sterilization under pressure, or burning.
4. Terminal disinfection: Thorough cleaning.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Search for history of exposure to infected animals or contaminated animal products and trace to place of origin; if in a manufacturing plant, inspect for adequacy of preventive measures outlined in 9A above.
8. Specific treatment: Prompt treatment with penicillin, tetracycline, or other broad spectrum antibiotic; supplementary sulfadiazine may be used in severe cases.

- C. Epidemic measures:** The occasional epidemics in man in the United States are local outbreaks among employees who work with animal products, especially goat hair. In rural populations where anthrax of animals is common, outbreaks have been related to butchering of infected cattle or as an occupational hazard of animal husbandry.
- D. International measures:** Sterilization of imported bone meal before use as animal feed. Sterilization of hair imported for use in shaving brushes; disinfection of wool and other products when desirable and practical, formaldehyde being commonly used.
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ARTHROPOD-BORNE VIRAL INFECTIONS

A Summary

A large number of arthropod-borne viruses produce disease in man. Clinical manifestations vary so greatly that the diseases ordinarily are distinguishable only by specific laboratory tests. Some are predominantly an encephalitis or the aseptic meningitis syndrome, others a febrile disease without involvement of the central nervous system, and still others predominantly systemic febrile illnesses associated occasionally with encephalitis or the aseptic meningitis syndrome. Several are characterized by occasional or predominantly hemorrhagic manifestations, one with severe liver damage and jaundice.

Most of these diseases are zoonoses, accidentally acquired by man through an arthropod vector, with man an unimportant host in the cycle. In the presence of a suitable vector a few may become epidemic, with man the principal source of vector infection. Still others are not at present recognized as basically dependent on an animal-arthropod cycle. Most of the viruses concerned are mosquito-borne, several are tick-borne, 2 are known to be phlebotomus-borne, and 1 is questionably transmitted by a mite. Laboratory infections occur.

Many common epidemiologic factors in transmission cycles, principally in relation to the vector, are of importance in respect to methods of control. Consequently, the several diseases are arranged in four groups: mosquito-borne, tick-borne, phlebotomus-borne, and 1 possibly mite-borne. Nevertheless, differences in vertebrate hosts, in geographic distribution, in availability of vaccine and other features require an individual consideration of epidemiology and control even for those with common vectors.

Certain diseases, therefore, are dealt with independently in alphabetic order in various sections of this book; these are Colorado Tick Fever, Dengue Fever, Phlebotomus Fever, Rift Valley Fever, and Yellow Fever. A limited number of the others are presented in detail as type examples in the 3 sections immediately following, namely, Arthropod-Borne Viral Encephalitides, Arthropod-Borne Viral Fevers, and Arthropod-Borne Viral Hemorrhagic Fevers.

Approximately 75 known viruses are readily classified by hemagglutination or complement-fixation into antigenic groups, of which A and B are the largest and best known. Both contain agents predominantly causing encephalitis and agents predominantly causing other febrile illnesses. Group A includes only mosquito-borne viruses, group B both mosquito- and tick-borne agents. Group C and 6 other small groups (2 to 5 viruses) for the most part are agents unassociated with disease. The 30 presently recognized agents for which no common antigens have been demonstrated remain in a miscellaneous category. All associated with human disease are listed below by type of vector reasonably established or suspected, predominant character of recognized disease and geographical area where found.

Virus Group	Name of Virus	Vector	Disease	Where Found
Group A	Chikungunya	Mosquito	Fever, Hem. fever	Africa, Thailand, p. 33
	Eastern equine	Mosquito	Encephalitis	See p. 30
	Mayaro	Mosquito	Fever	S. America, p. 33
	Scutiger Forest	Mosquito	Fever	Africa
	Uruba	Mosquito (?)	Fever	S. America
	Venezuelan equine	Mosquito	Fever, encephalitis (?)	S. America, p. 33
	Western equine	Mosquito	Encephalitis	See p. 30
	Dengue 1, 2	Mosquito	Fever	See p. 62
	Dengue 3, 4	Mosquito	Hemorrhagic fever	See p. 34
	(Philippine hemorrhagic fever)			
Group B	Diphasic meningo-encephalitis	Tick	Encephalitis	See p. 32
	Ilheus	Mosquito	Fever, encephalitis (?)	S. America
	Japanese B	Mosquito	Encephalitis	See p. 30
	Kanamar Forest	Tick	Hemorrhagic fever	See p. 36
	Louping ill	Tick	Encephalitis	See p. 32
	Murray Valley	Mosquito	Encephalitis	See p. 30
	Nairobi sheep	Tick	(?)	Africa
	Onk hemorrhagic	Tick	Hemorrhagic fever	See p. 36
	Powassan	Unknown	Encephalitis	Canada
	Russian spring summer	Tick	Encephalitis	See p. 32
	Spondweni	Mosquito	Fever	Africa
	St. Louis	Mosquito	Encephalitis	See p. 30

Virus Group	Name of Virus	Vector	Disease	Where Found
Group C	Uganda S	Mosquito	Fever	Africa
	Wesselsbron	Mosquito	Fever	Africa
	West Nile	Mosquito	Fever	See p. 33
	Yellow fever	Mosquito	Hemorrhagic fever	See p. 223
	Zika	Mosquito	Fever	Africa
Group C	Caraparu	Mosquito	Fever	S. America
	Marituba	Mosquito	Fever	S. America
	Oriboca	Mosquito (?)	Fever	S. America
Guama Group	Guama (BeAn 277)	Mosquito (?)	Fever	S. America
	Catu (BeH 151 (?))	Mosquito (?)	Fever	S. America
Bwamba Group	Bwamba	Mosquito	Fever	Africa, p. 33
Bunyamwera Group	Bunyamwera	Mosquito	Fever	Africa, p. 33
	Germiston	Mosquito	Fever	Africa
	Ilesha	Unknown	Fever	Africa
California Group	California	Mosquito	Encephalitis	United States
Ungrouped	Colorado tick fever	Tick	Fever	See p. 58
	Crimean hemorrhagic fever	Tick	Hemorrhagic fever	See p. 35
	Epidemic hemorrhagic fever	Mite (?)	Hemorrhagic fever	See p. 37
	Phlebotomus fever, Naples type	Phlebotomus	Fever	See p. 126
	Phlebotomus fever, Sicilian type	Phlebotomus	Fever	See p. 126
	Quaranfil	Tick	Fever	Africa
	Rift Valley	Mosquito	Fever	See p. 152

ARTHIPOD-BORNE VIRAL ENCEPHALITIDES

I. Mosquito-Borne

A. EASTERN EQUINE, WESTERN EQUINE, JAPANESE B, MURRAY VALLEY, AND ST. LOUIS TYPES

1. **Identification**—A group of acute inflammatory diseases of short duration, involving parts of the brain, spinal cord, and meninges. Signs and symptoms are similar but with variation in severity and rate of progress. Mild cases may present the aseptic meningitis syndrome. Severe infections usually have acute onset, high fever, meningeal signs, stupor, disorientation, coma, spasticity, tremors, occasionally convulsions in infants, and spastic but rarely flaccid paralysis. Fatality ranges from 5 to 60%, that of Japanese B and Eastern equine types being highest. Permanent sequelae are rare except in infants; no parkinsonism. Mild leucocytosis is usual; leucocytes in spinal fluid from 50 to 200 per ccm, occasionally 1,000 or more in infants.

Specific identification is by demonstrated rise in antibody between early and late specimens of serum, by neutralization, complement fixation, and hemagglutination inhibition; group reactions may occur. Virus may be isolated from the brain of fatal cases; histopathological changes are not specific for individual viruses.

These diseases require differentiation from encephalitic and nonparalytic forms of poliomyelitis, rabies, mumps meningoencephalitis, lymphocytic choriomeningitis, aseptic meningitis syndrome due to ECHO and Coxsackie viruses, herpes encephalitis, postvaccinal or postinfection encephalitides, bacterial, protozoal, leptospiral and fungal meningitides or encephalitides; also the von Economo type of encephalitis (encephalitis lethargica) of unknown etiology, a disease of frequent occurrence just before and after 1920 but now rarely reported. The conditions listed are to be reported to official health agencies under the appropriate disease: or as Encephalitis, other forms, or as Meningitis, other forms, or as Aseptic Meningitis Syndrome, with etiology or clinical type specified when known.

2. **Occurrence**—Eastern equine encephalitis is recognized in eastern United States and Canada and scattered areas of Central and South America, also reported from Luzon, P. I., and in eastern Slovakia; Western equine in western United States and Canada, scattered areas further east and in South America, isolation also reported from Slovakia; Japanese B in western Pacific Islands from Japan to Guam, many eastern areas of Asia from Korea to Singapore and India; Murray Valley in parts of Australia and New Guinea; and St. Louis in most parts of the United States except the Northeast, also in Trinidad and Panama. Disease occurs in temperate latitudes in summer and early fall. Commonly limited to areas and years of high temperature and many mosquitoes; has persisted endemically in hot, irrigated valley regions; irregularly noted in dry farming areas. May also be seasonal in tropical countries depending on rainfall and vector population.
3. **Infectious agents**—Each disease is caused by a specific virus: Eastern equine, Western equine, Japanese B, Murray Valley and St. Louis viruses. The first 2 are in group A, the last 3 in group B.
4. **Reservoir and source of infection**—True reservoir or means of winter carry-over unknown, possibly bird or surviving adult mosquito. Source of infection for man is an infective mosquito.
5. **Mode of transmission**—By the bite of mosquitoes. Mosquitoes usually acquire the infection from wild birds but pigs are of some importance

for Japanese B; for others occasionally a mammal, such as the horse. Virus is not demonstrable in the blood of man after onset of disease and probably never present in sufficient amount to infect a mosquito. Horses develop active disease with the 2 equine viruses and Japanese B, but viremia rarely is present for long periods or in high titer, therefore uncommon sources of mosquito infection. Most important vectors are: Eastern equine, probably *Culiseta melanura*; Western equine, *Culex tarsalis*; Japanese B, *Culex tritaeniorhynchus* and *Culex gelidus*; Murray Valley, probably *Culex annulirostris*; St. Louis, *Culex tarsalis* and *Culex pipiens-quinquefasciatus* complex.

6. **Incubation period**—Usually 5 to 15 days.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Mosquito remains infective for life. Viremia in birds usually lasts 2 to 5 days.
8. **Susceptibility and resistance**—Susceptibility to clinical disease usually highest in infancy and old age but varies with type; inapparent or undiagnosed infection is more common at other ages. Infection results in homologous immunity. In highly endemic areas adults are largely immune to local strains by reason of mild and inapparent infections, and susceptibles are mainly children.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Destruction of larvae and elimination of breeding places of known or suspected vector mosquitoes.
 2. Killing mosquitoes by space and residual spraying of human habitations.
 3. Screening of sleeping and living quarters; use of mosquito bed-nets.
 4. Avoid exposure to mosquitoes during hours of biting, or use repellents.
 5. Education of the public as to mode of spread and control.
 6. Immunization of persons at great risk (laboratory workers and others) with experimental or commercially prepared equine formalinized vaccines; not recommended for general use.
 7. Passive protection of accidentally exposed laboratory workers by human or animal immune serum.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Case report obligatory in most states of the United States and in some other countries, Class 2A (p. 5).
 2. Isolation: None; virus not usually found in blood, secretions, or discharges during clinical manifestations.
 3. Concurrent disinfection: None.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Search for missed cases and presence of vector mosquitoes; primarily a community problem (see 9C).
 8. Specific treatment: None.
 - C. **Epidemic measures:** Identification of the disease among horses or birds and recognition of human cases in the community has epidemiological value to indicate frequency of infection and areas involved.
 - D. **International measures:** Insecticide spray of airplanes arriving from recognized areas of prevalence.

II. Tick-Borne

A. RUSSIAN SPRING-SUMMER, LOUPING ILL, AND DIPHASIC MENINGOENCEPHALITIS

1. **Identification**—A group of diseases clinically resembling mosquito-borne encephalides except that Russian spring-summer type often is associated with flaccid paralysis, particularly of the shoulder girdle and frequently leaving residual manifestations. Diphasic meningoencephalitis (diphasic milk fever) has a longer course, averaging 3 weeks. The initial febrile stage is unassociated with symptoms referable to the central nervous system; a second phase of fever and meningoencephalitis follows 4 to 10 days after apparent recovery. Fatality and severe residua are less than for the Russian spring-summer disease. Louping ill in man also has a diphasic pattern and is relatively mild.

Specimen identification is by serological tests or by isolation of virus from blood during acute phase of illness. Serological tests cannot be expected to differentiate between members of this group, but do distinguish them from most other similar diseases.

2. **Occurrence**—Disease from this virus complex has spotty distribution over much of USSR, other parts of Eastern and Central Europe, Scandinavia, Scotland, and the north of England. In general the spring-summer type has a more eastern or Asian distribution, diphasic meningoencephalitis predominates in Europe, with louping ill present in Scotland and England. Infectious agents of the same antigenic complex have been found in Canada, Malaya and India. Disease in man has not been recognized in Malaya and the disease in India is described as hemorrhagic fever. Seasonal incidence depends on activity of the tick, *Ixodes persulcatus*, usually spring and early summer, while *I. ricinus* continues into late summer or early autumn. Areas of highest incidence are where man has the most intimate association with the largest numbers of infected ticks. This is generally in rural or forest areas but also in urban populations. Small local epidemics of diphasic meningoencephalitis have occurred among persons consuming raw goat or sheep milk, thus the name diphasic milk fever. Laboratory infections have been common, a number with serious sequelae and some fatal.
3. **Infectious agents**—Minor antigenic differences exist, but viruses in these diseases are closely related to form a complex, usually designated the Russian spring-summer louping ill group. All are in group B.
4. **Reservoir and source of infection**—The tick appears to be the true reservoir; transovarian passage of virus has been demonstrated. Rodents, sometimes other mammals and birds, rarely man, give rise to tick infection. The source of infection for man is an infective adult tick.
5. **Mode of transmission**—By the bite of infective ticks, or consumption of milk from infected animals. *I. persulcatus* is the principal vector in eastern USSR and *I. ricinus* in western USSR and other parts of Europe; the latter is also the vector of louping ill of sheep in Scotland. Certain *Haemaphysalis* species also are involved in some regions. Ticks in the larval stage usually obtain virus by feeding on rodents, sometimes other mammals and birds. Man may be responsible for occasional infection of adult ticks.
6. **Incubation period**—Usually 7 to 14 days.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. A tick infected at any stage remains infective for life and its progeny also may transmit. Viremia in a variety of vertebrates may last for several days; in man up to a week or 10 days.

8. **Susceptibility and resistance**—Both sexes and all ages are susceptible, but the age pattern varies widely in different regions as influenced by exposure to ticks, consumption of milk from an infected animal, or previously acquired immunity. Infection, whether inapparent or overt, leads to immunity. Suitable vaccines produce immunity.
9. **Methods of control**—
- A. Preventive measures:**
1. See Rocky Mountain Spotted Fever, p. 160, for preventive measures against ticks.
 2. Formalinized virus vaccines have been used extensively in the USSR with reported safety and effectiveness.
 3. Boiling or pasteurizing of milk of animals of susceptible species.
- B. Control of patient, contacts, and the immediate environment:**
1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3B (p. 5).
 2. Isolation: None, if patient is tick-free.
 3. Concurrent disinfection: None.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Search for missed cases and presence of tick vectors or animals excreting virus in milk.
 8. Specific treatment: None.
- C. Epidemic measures:** See Rocky Mountain Spotted Fever, p. 160.
- D. International measures:** None.

ARTHROPOD-BORNE VIRAL FEVERS

I. Mosquito-Borne

A. WEST NILE, VENEZUELAN EQUINE, CHIKUNGUNYA, MAYARO, BWAMBA, AND BUNYAMWERA FEVERS

1. **Identification**—A group of acute febrile dengue-like illnesses of a week or less, and seldom fatal. Usual onset is with headache, malaise, and occasionally nausea and vomiting; generally some conjunctivitis. Temperature may or may not be of the saddleback type, with rashes in many patients with West Nile and Chikungunya infections. Meningoencephalitis is an occasional feature of West Nile infections and hemorrhages with shock in Chikungunya.
- Serological tests are useful in differentiating from other fevers of viral or unknown origin, but Chikungunya and Mayaro virus infections are difficult to distinguish from one another. Specific diagnosis also possible by virus isolation from blood during febrile period in many of these infections. Venezuelan also recovered from nasopharyngeal washings.
2. **Occurrence**—West Nile fever occurs in Egypt, Israel, India, and probably is widespread in a number of parts of Africa. Chikungunya has been encountered in Africa and Thailand. Bwamba and Bunyamwera fevers thus far have been identified only in a few areas in Africa. Venezuelan equine and Mayaro fevers occur in tropical South America. Seasonal

incidence depends on conditions for high vector prevalence. Recognized occurrence is primarily rural.

3. **Infectious agents**—Each disease is due to an independent virus of the same name as the disease. West Nile virus is in group B, and the closely related Chikungunya and Mayaro viruses in group A.
4. **Reservoir and source of infection**—Reservoir is unknown. The source of human infection is an infective mosquito.
5. **Mode of transmission**—By the bite of a mosquito. For West Nile, mosquito infected most frequently from wild birds; for other viruses possibly from man. *Culex univittatus* is the vector of West Nile virus in Egypt, *Culex pipiens molestus* in Israel, *Aedes aegypti* for Chikungunya, *Aedes sp.* for Bunyamwera fever and *Mansonia venezuelensis* for Mayaro.
6. **Incubation period**—Usually 3 to 12 days.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Infective mosquitoes probably transmit the virus throughout life.
8. **Susceptibility and resistance**—Susceptibility appears to be general, in both sexes and throughout life. Inapparent infections and mild undiagnosed disease are relatively common. Infection leads to immunity and susceptibles in highly endemic areas are mainly among young children.
9. **Methods of control**—See Mosquito-Borne Encephalitides, p. 31.

B. DENGUE FEVER, see p. 62

C. RIFT VALLEY FEVER, see p. 152

II. Tick-Borne

A. COLORADO TICK FEVER, VIRAL, see p. 58

III. Phlebotomus-Borne

A. PHLEBOTOMUS FEVER, see p. 126

ARTHROPOD-BORNE VIRAL HEMORRHAGIC FEVERS

I. Mosquito-Borne

A. PHILIPPINE AND THAILAND HEMORRHAGIC FEVERS

1. **Identification**—A group of epidemic acute hemorrhagic fevers recognized so far principally in children under 5 years of age. Sudden onset with high fever, prostration, headache, malaise, frequently nausea and vomiting. Conjunctivitis, epistaxis, and abdominal pain soon appear, along with an early petechial rash first noted on the extremities and not infrequently involving face and trunk but not axillae or chest. Purpuric lesions may appear early and extensive ecchymoses later. Many patients have gastrointestinal hemorrhage and serious shock about the second to fifth day, at which time blood platelets are reduced and bleeding time prolonged. Usually no leucopenia or leucocytosis. Duration is usually 5 to 8 days with prompt uncomplicated recovery. Renal hemorrhages and albuminuria are rare or inconspicuous. Fatality is about 10%, with death mainly during period of shock.

Serologic tests give rises in titer with the responsible viruses and others closely related. Virus may be isolated from blood during acute febrile stage.

2. **Occurrence**—All recognized outbreaks have been in urban areas of Philippine Islands and in Thailand. Major outbreaks in Manila and Bangkok,

but smaller urban areas also involved. Occurrence is limited to rainy seasons and areas of high *A. aegypti* prevalence. Not observed where only *Aedes albopictus* is present.

3. **Infectious agents**—Viruses presently classed as dengue types 2, 3 and 4 appear to cause the disease in the Philippines. In Thailand dengue types 1 and 2 and Chikungunya appear responsible.
4. **Reservoir and source of infection**—Reservoir unknown. Immediate source of infection is probably an infective *A. aegypti*.
5. **Mode of transmission**—Probably by the bite of an infective *A. aegypti* mosquito, with infection acquired from man.
6. **Incubation period**—Unknown.
7. **Period of communicability**—Under natural conditions not believed directly transmissible from man to man.
8. **Susceptibility and resistance**—Modal age of attack in epidemics thus far recognized is about 3 to 5 years, with range from 4 months to young adults. Prevalence of dengue antibodies in the general population is high in older children and in adults; little known regarding immunity pattern in man. Many mild and inapparent infections recognized in Philippines by serologic tests.
9. **Methods of control**—See Dengue Fever, p. 62 and Yellow Fever, p. 223.

B. YELLOW FEVER, see p. 223

II. Tick-Borne

A. CRIMEAN HEMORRHAGIC FEVER

1. **Identification**—Onset sudden with fever, malaise, weakness, irritability, headache, severe pain in limbs and loins and marked anorexia. Vomiting, abdominal pain, and diarrhea occur occasionally. Early development of flush on face and chest, and injection of conjunctivae. Associated with hemorrhagic enanthem of soft palate, uvula and pharynx, and a fine petechial rash spreading from chest and abdomen to the body generally; occasional large purpuric areas. Some bleeding from gums, nose, lungs, uterus and intestine; in large amount only in serious or fatal cases. Hematuria and albuminuria slight or absent. Fever constantly elevated for 5 to 12 days, falling by lysis with prolonged convalescence. Leucopenia with marked neutropenia; relative lymphocytosis but absolute lymphopenia and eosinophilia. Thrombocytopenia present but bleeding time is normal. Reported fatality ranges from 2 to 15%.

Specific diagnosis is possible by virus isolation from blood during acute phase and by serological tests.
2. **Occurrence**—Recognized in the steppe regions of Western Crimea and on the Kerch peninsula. Most patients are agricultural workers in fallow lands where hares are numerous. Seasonal occurrence from June to September, the period of vector activity.
3. **Infectious agent**—The virus of Crimean hemorrhagic fever, an ungrouped viral agent.
4. **Reservoir and source of infection**—Reservoir in nature is hares, possibly birds, and a *Hyalomma* tick. Immature ticks are believed to acquire infection from animal hosts; after molt to an adult form they are the source of infection for man.
5. **Mode of transmission**—By the bite of adult infective *Hyalomma marginatum* ticks.

6. **Incubation period**—7 to 12 days.

7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Infected tick remains so for life.

8. **Susceptibility and resistance**—Immunity is known to last at least 1 year.

9. **Methods of control**—

A. Preventive measures:

1. See Rocky Mountain Spotted Fever, p. 160, for preventive measures against ticks.

2. No available vaccine.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3B, p. 5.

2. Isolation: None, if patient is tick-free.

3. Concurrent disinfection: None.

4. Terminal disinfection: None.

5. Quarantine: None.

6. Immunization: None.

7. Investigation of contacts and source of infection: Search for missed cases and presence of vectors and hares.

8. Specific treatment: Convalescent serum reported useful.

C. Epidemic measures: See Rocky Mountain Spotted Fever, p. 160.

D. International measures: None.

B. OMSK HEMORRHAGIC FEVER AND KYASANUR FOREST DISEASE

1. **Identification**—These 2 diseases have marked similarities. Onset is sudden with headache, fever, pain in low back and limbs, and severe prostration; often associated with conjunctivitis, diarrhea, and vomiting by the third to fourth day. A papulo-vesicular eruption on the soft palate is an important diagnostic sign. No involvement of the central nervous system. Severe cases are associated with hemorrhages but no cutaneous rash. Bleeding may occur from gums, nose, gastrointestinal tract, uterus, and lungs but rarely from the kidney, sometimes for many days and when serious, results in shock and death. Estimated fatality is from 1 to 10%. Leucopenia and thrombocytopenia are marked. The febrile period ranges from 5 days to 2 weeks, sometimes with a secondary rise in the third week. Recovery and convalescence tend to be slow and prolonged.

Diagnosis is by isolation of virus from blood, possibly as long as 10 days following onset, or by serological tests. Not readily differentiated serologically from Russian spring-summer encephalitis or louping ill.

2. **Occurrence**—Recognized in the Kyasanur Forest of Shimoga District, Mysore State, India, principally in young adult males exposed in the forest during the dry season from January to June. Omsk type occurs in rural workers and children exposed to infected ticks in the steppe regions of Omsk Oblast in Siberia. The seasonal occurrence in each area is that of the vector. Laboratory infections are common with both viruses.

3. **Infectious agents**—Viruses of these two diseases are closely related. The agents belong to group B, are of the Russian spring-summer louping ill complex, and similar antigenically to other members.

4. **Reservoir and source of infection**—Reservoir in nature, probably monkeys in Kyasanur Forest disease; in Omsk disease, rodents and muskrats, together with the tick since transovarian passage has been reported for other viruses of this complex. The larval stages of ticks appear to be the principal source of infection for monkeys and man.
5. **Mode of transmission**—By the bite of infective ticks: *Dermacentor pictus* and *D. marginatus* in the Omsk type and probably *Haemaphysalis spinigera* in Kyasanur Forest disease.
6. **Incubation period**—Usually 3 to 8 days.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Infected tick remains so for life.
8. **Susceptibility and resistance**—All ages and sexes are probably susceptible; previous infection leads to immunity.
9. **Methods of control**—See Tick-Borne Encephalitis p. 32, and Rocky Mountain Spotted Fever p. 160. A formalinized mouse-brain virus vaccine has been reported effective.

III. Mite-Borne (Possible)

A. EPIDEMIC HEMORRHAGIC FEVER

1. **Identification**—An acute infectious disease characterized by fever of 3 to 6 days' duration, conjunctival injection, prostration, anorexia, vomiting, hemorrhagic manifestations which begin about the third day, proteinuria about the fourth day, and hypotension about the fifth; renal abnormalities continue for several weeks. About one-fourth of cases show an alarming hypotension and the majority of deaths (fatality about 6%) occur during shock. Convalescence is usually rapid during the third week. Synonym: Hemorrhagic Nephroso-nephritis.
Specific laboratory diagnostic tests are not available; clinical laboratory findings such as proteinuria, leucocytosis, thrombocytopenia, and elevated nonprotein nitrogen assist in establishing the diagnosis.
2. **Occurrence**—The disease was encountered in Korea in the vicinity of the 38th parallel among United Nations troops in 1951. Earlier Japanese and Russian experience in Manchuria and Siberia along the Amur River and its tributaries indicates that women and children acquire the malady as well as men. Recently recognized also in European USSR in the Yaroslavl region north of Moscow. Two seasonal outbreaks, in May and June and in October and November, account for most cases in Korea; a few occur throughout the year. The majority are isolated events but outbreaks are observed involving 5 to 20 persons within a small area, all apparently acquired at the same time and place.
3. **Infectious agent**—Relatively little known; Berkefeld and Seitz filtrates of infectious human materials induce hemorrhagic fever in experimentally inoculated volunteers. The agent has not been established in laboratory animals.
4. **Reservoir and source of infection**—Assumed to be maintained in nature by a cycle involving some terrestrial arthropod and some rodent, with man only an accidental host.
5. **Mode of transmission**—Unknown; epidemiologic observations in Korea suggest an analogy to scrub typhus and implicate a nonflying arthropod vector of limited mobility; trombiculid mites are a likely suggestion.
6. **Incubation period**—Usually 12 to 16 days but varying from 9 to 35.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man.

8. Susceptibility and resistance—Newcomers to endemic areas are uniformly susceptible; indigenous populations probably have some acquired resistance. Mild or inapparent infections are suspected but remain unproved in the absence of specific diagnostic tests. Second attacks have not been observed.

9. Methods of control—

A. Preventive measures: Lacking adequate information of etiologic agent and mode of transmission, preventive measures are those of diseases with an arthropod vector and a rodent host; in Korea since the summer of 1952 essentially as for Scrub Typhus (see pp. 168, 169).

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3A (p. 5).

2. Isolation: None.

3. Concurrent disinfection: None.

4. Terminal disinfection: None.

5. Quarantine: None.

6. Immunization of contacts: None.

7. Investigation of source of infection: None.

8. Specific treatment: None.

C. Epidemic measures: Since outbreaks apparently result from simultaneous infection of groups, control measures are not applicable to the episode. For measures in an endemic area, see 9A above, and Scrub Typhus.

D. International measures: None.

ASCARIASIS

1. Identification—A common chronic intestinal infection by a nematode. Symptoms variable and often vague or absent; heavy infection may cause digestive disturbances, abdominal pain, vomiting, restlessness, and disturbed sleep. Live worms passed in stools or vomited are frequently the first sign of infection. Ordinarily a mild infection, but heavy infections may result in serious complications, with deaths not uncommon among children in tropical countries.

Identification of eggs in feces is the usual method of diagnosis.

2. Occurrence—Common world-wide infection, most frequent in moist tropical countries where prevalence may exceed 50% of a population. Children of preschool and early school age are more frequently and more heavily infected than older children and adults. In the United States the disease is more prevalent in the South.

3. Infectious agents—*Ascaris lumbricoides*, the large intestinal round worm of man; rarely the canine or feline variety. After ingestion, embryonated eggs hatch in intestinal canal, larvae penetrate wall, and reach liver and lungs by way of lymphatic and circulatory systems. Most larvae reaching the lungs pass into air passages, ascend bronchi, are swallowed, and eventually reach the small intestine where they grow to maturity and mate. Eggs are discharged from gravid female and leave infected person with feces.

4. Reservoir and source of infection—The reservoir is an infected person discharging eggs in feces. The immediate source of infection is soil containing embryonated eggs, in and about houses where facilities for sanitary disposal of human excreta are lacking or not used.

5. **Mode of transmission**—By direct or indirect transmission of embryonated eggs from soil or other contaminated material to mouth; embryonation requires about a month after passage of the egg. Salads and other foods eaten raw are vehicles. Contaminated soil may be carried long distances on feet or footwear into houses and conveyances, also as dust.
6. **Incubation period**—Worms reach maturity about 2 months after ingestion.
7. **Period of communicability**—As long as mature fertilized female worms live in intestine. Most adult worms live less than 6 months; maximum life under 1 year. The female produces about 200,000 eggs a day. Embryonated eggs under favorable conditions remain viable in soil for months and even for years.
8. **Susceptibility and resistance**—Susceptibility is general.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Provision of adequate facilities for proper disposal of feces and prevention of soil contamination in areas immediately adjacent to houses, particularly in play areas of children.
 2. In rural areas, construction of privies in such manner as to obviate dissemination of ascarid eggs through overflow, drainage, or similar circumstance. Composting is practiced to advantage where such facilities are lacking.
 3. Education of all persons, particularly children, to use toilet facilities, and to wash hands after defecating.
 4. Encouragement of satisfactory hygienic habits on the part of children, especially the practice of washing hands before handling food, and the danger of eating food dropped on the floor.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
 2. Isolation: None.
 3. Concurrent disinfection: Sanitary disposal of feces.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Individual and environmental sources of infection should be sought, particularly in persons and premises of family.
 8. Specific treatment: Piperazine derivatives.
 - C. **Epidemic measures:** Surveys for prevalence in highly endemic areas, education in sanitation of environment and in personal hygiene, and provision of treatment facilities.
 - D. **International measures:** None.

ASEPTIC MENINGITIS SYNDROME

1. **Identification**—A group of diseases of diverse etiology presenting a common clinical syndrome. A relatively short and benign illness characterized by an acute onset and obvious signs and symptoms of meningeal

irritation unrelated to parameningeal infection or generalized disease which might give rise to meningitis as a secondary manifestation. There is no paralysis and bacteria cannot be recovered from the cerebrospinal fluid which invariably contains increased numbers of white cells and an elevated protein content. Lymphocytes predominate in the spinal fluid but polymorphonuclear cells are not infrequent early in the disease. Epidemiologically, often occurs in the absence of other types of meningitis. Cases have been described with respiratory or gastrointestinal involvement or with a morbilliform rash. Synonyms: **Non-bacterial Meningitis, Viral Meningitis, Serous Meningitis.**

Specific diagnosis is only by laboratory means, either by isolation of the infectious agent or by demonstrated rise in specific antibody in convalescent as compared with acute phase serums. Using present day laboratory procedures under optimal conditions, the infectious agent can be determined in about 75% of all cases of aseptic meningitis.

Chronic meningitis is usually the result of meningeal or cerebral tuberculosis or inadequately treated bacterial or mycotic disease, particularly cryptococcosis. Chronic viral meningitis is unknown.

2. **Occurrence**—World-wide distribution in urban populations. Occurs sporadically except that Coxsackie, ICHO and some other viruses produce disease of this nature reaching epidemic levels. Actual incidence is unknown but may be greater than is generally recognized.
3. **Infectious agents**—Caused by a wide variety of infectious agents; most viruses are assumed potentially capable of producing this syndrome. The commonest are Group B Coxsackie (p. 131), mumps (p. 118), ICHO (p. 101, par. 3), lymphocytic choriomeningitis (p. 108), and poliomyelitis virus (p. 137). Less frequently implicated are the virus of herpes simplex, arthropod borne viruses (p. 27), and the leptospiras (p. 105).
4. 5. 6. 7. 8. **Reservoir and source of infection, Mode of transmission, Incubation period, Period of communicability, Susceptibility and resistance**—These vary according to the specific infectious agent (see the particular disease).

9. **Methods of control**—

A. Preventive measures: Dependent upon etiology (see specific diseases).

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas (USA); in many countries not a reportable disease, Class 3B (pp. 5, 6). If confirmed by laboratory means, specify the infectious agent; otherwise, as cause undetermined.
2. Isolation: Since specific diagnosis can only be made by laboratory methods and will not be certain until patient has recovered, isolation of all cases during febrile period is indicated.
3. Concurrent disinfection: Of eating and drinking utensils and articles soiled by secretions and excretions of patient.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: See particular disease.
7. Investigation of contacts and source of infection: Not profitable.
8. Specific treatment: See particular disease.

C. Epidemic measures: See specific disease.

D. International measures: None.

BARTONELLOSIS

1. **Identification**—An illness occurring in 2 stages, an interval of weeks to months usually separating an initial acute febrile period and an eruptive period. The febrile period, or Oroya fever stage, is characterized by irregular fever, rapidly developing severe anemia, pain in bones and joints, and lymphadenopathy. The eruptive or Verruga Peruana stage may merge with the febrile stage; an intervening quiescent period is usual. The eruption is in crops and consists of papules or nodules resembling hemangiomas, sometimes with many small lesions, sometimes with a few tumor-like subepithelial nodules. The fatality of untreated Oroya fever ranges from 10 to 40%; Verruga Peruana is prolonged but with few deaths. Synonyms: Oroya Fever, Verruga Peruana.

Laboratory diagnosis is by demonstration of the infectious agent in stained smears of the blood during the acute stage, in sections of skin lesions during the eruptive stage, or by blood culture during either stage.

2. **Occurrence**—Limited to certain altitudes in mountain valleys of Peru, Ecuador, and southwest Colombia where vector is present. No special predilection for age, race, or sex.
3. **Infectious agent**—*Bartonella bacilliformis*.
4. **Reservoir and source of infection**—Reservoir is an infected person with the infectious agent present in the blood. The source of infection is a sandfly.
5. **Mode of transmission**—By bite of sandflies of the genus *Phlebotomus*. Vector species not identified for all areas; *P. verrugarum* is important in Peru. Under natural conditions, not directly transmissible from man to man.
6. **Incubation period**—Usually 16 to 22 days but occasionally 3 to 4 months.
7. **Period of communicability**—Prolonged; the infectious agent may be demonstrated by blood culture weeks before and up to several years after actual illness. Duration of infectivity of sandfly, unknown.
8. **Susceptibility and resistance**—Susceptibility is general but the disease is milder in children than adults. Inapparent infections and the carrier state are known. Recovery from an untreated attack of Oroya fever almost invariably gives permanent immunity to this form. The Verruga Peruana stage may recur.

9. Methods of control—

A. Preventive measures:

1. The control of sandflies. The measures are those listed under Cutaneous Leishmaniasis, 9A and 9C, pp. 101, 102.
2. Avoid known endemic areas after sundown; otherwise carefully apply insect repellent to exposed parts of the body.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3B (pp. 5, 6).
2. Isolation: None. The infected individual should be protected from bites of *Phlebotomus* (see 9A).
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.

7. Investigation of contacts and source of infection: Identification of sandflies, particularly in localities where the infected person was exposed after sundown during preceding 3 to 8 weeks.
 8. Specific treatment: Penicillin, streptomycin, chloramphenicol and tetracyclines are all effective in reducing the fever and the bacteremia. Blood transfusion is useful in the phase of acute anemia.
- C. *Epidemic measures*: Intensification of case finding and systematic spraying of houses with DDT for residual effect.
- D. *International measures*: None.

BLASTOMYCOSIS, NORTH AMERICAN

1. **Identification**—A chronic fungus disease, occasionally with acute onset, occurring in two clinical forms: (a) Cutaneous blastomycosis—A chronic granulomatous disease characterized by a papule which ulcerates and spreads slowly and peripherally for months or years, leaving an irregular crusted ulcer with a granulomatous base and an elevated papilliform to verrucous border containing minute abscesses. The center of the ulcer heals with a thin scar. The lesions are usually on exposed parts of the body such as face, hands, wrists, feet and ankles. Primary cutaneous blastomycosis is often chronic with little tendency to become generalized. Cutaneous blastomycosis ordinarily is a local manifestation of existing systemic disease. (b) Systemic blastomycosis—A chronic granulomatous mycosis, primarily of the lungs; begins with fever and symptoms of upper respiratory infection resembling influenza; progresses gradually with fever, loss of weight, cachexia with cough, purulent sputum, and abscesses in subcutaneous tissues, bones, central nervous system, and reproductive and other visceral organs; highly fatal if untreated.
Direct microscopic examination of unstained smears of sputum and materials from lesions shows characteristic budding forms of the fungus which can be cultured. A rise and fall in complement fixation titer is diagnostic if histoplasmosis and other mycoses can be excluded.
2. **Occurrence**—Uncommon, occurring sporadically in central and south-eastern United States, in Central America, and in Canada. Occurs at all ages, but two-thirds of cases are between 15 and 45 years. Males are infected 7 times more frequently than females. Infection of dogs is reported with increasing frequency; horses occasionally.
3. **Infectious agent**—*Blastomyces dermatitidis*, a dimorphic fungus which grows as a yeast in the tissues and in culture at 37° C. on blood or other enriched media, and on simple media as a mold at 30° C.
4. **Reservoir and source of infection**—Reservoir probably is soil; the immediate source of infection is probably spore-laden dust.
5. **Mode of transmission**—Probably the resistant spores typical of the mold, i.e., the saprophytic growth phase of the fungus, are inhaled in spore-laden dust or introduced through wounds or abrasions.
6. **Incubation period**—Probably a few weeks or less.
7. **Period of communicability**—Unknown. Under natural conditions not directly transmissible from man or animals to man. Contamination of the environment with resultant conversion of the fungus from the parasitic growth phase to the saprophytic disseminative mold phase is potentially possible for duration of the disease.

8. **Susceptibility and resistance**—Unknown. Inapparent pulmonary infections are probable but of undetermined frequency. No information on immunity; rarity of the disease and of laboratory infections suggest man is relatively resistant to the disseminated fatal form of the disease.
9. **Methods of control**—
- A. Preventive measures:**
1. Diseased animals should be destroyed to avoid contamination of the environment.
- B. Control of patient, contacts, and the immediate environment:**
1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
 2. Isolation: None.
 3. Concurrent disinfection: Sputum, discharges, and all contaminated articles.
 4. Terminal disinfection: Thorough cleaning.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Not profitable.
 8. Specific treatment: Amphotericin B is the present drug of choice. Stilbamidine and 2-hydroxystilbamidine are effective in some cases but must be used with caution.
- C. Epidemic measures:** Not applicable, a sporadic disease.
- D. International measures:** None.
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BLASTOMYCOSIS, SOUTH AMERICAN

1. **Identification**—A highly fatal, chronic granulomatous mycosis with ulcerative lesions of skin and mucosa around mouth and nose, and in tongue, cheeks, and tonsils. Oral and periodontal lesions with loosening and loss of teeth are common. Other clinical forms include lymphatic involvement of the neck, with or without oral lesions, and infection of gastrointestinal tract, lungs, spleen, kidneys, pancreas, and other viscera. A keloidal form of the disease is recognized but its identity and etiology are controversial. Synonym: Paracoccidioidal Granuloma.
2. **Occurrence**—Endemic in South America, particularly rural Brazil. Highest incidence in adults aged 20 to 30 years; 10 times as common in males as in females.
3. **Infectious agent**—*Paracoccidioides brasiliensis* (*Blastomyces brasiliensis*), a dimorphic fungus.
4. **Reservoir and source of infection**—Reservoir presumably is wood, soil, or vegetation. Fungus demonstrated in apparently normal mouths, especially around teeth, probably represents incubation period of disease. The source of infection is probably wood, soil or spore-laden dust.
5. **Mode of transmission**—Presumably through contact with contaminated soil or vegetable materials.
6. **Incubation period**—Probably a few weeks or less.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—Unknown.

9. Methods of control—

A. *Preventive measures*: None.

B. *Control of patient, contacts, and the immediate environment*:

1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
2. Isolation: None.
3. Concurrent disinfection: Of discharges and contaminated articles.
4. Terminal disinfection: Thorough cleaning.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Not profitable.
8. Specific treatment: Sulfadiazine, sulfamerazine, and combinations of sulfonamides control the disease but treatment must be continued indefinitely to prevent relapse. Amphotericin B arrests spread of lesions promptly and is drug of choice.

C. *Epidemic measures*: Not applicable; a sporadic disease.

D. *International measures*: None.

BOTULISM

1. **Identification**—A highly fatal afebrile poisoning (not an infection) characterized by headache, weakness, constipation, oculomotor or other paralysis, and absence of diarrhea. Death by cardiac or respiratory paralysis occurs in about two-thirds of patients and usually within 3 to 7 days. Symptoms develop according to amount of toxin ingested in relation to body weight.

Biologic and toxicologic tests may confirm presence of bacterium or its toxin in suspected food or stomach contents.

Botulism is one of several varieties of food poisoning due to bacterial toxins. It is distinguished from Staphylococcal Food Poisoning (p. 80) and that due to the Welch bacillus, and from food poisoning by *Salmonella* (p. 162) in that the clinical manifestations relate primarily to the central nervous system rather than the gastrointestinal tract.

2. **Occurrence**—Sporadic and grouped cases occur in all countries and always in relation to some perishable food product which has been so prepared or preserved as to permit toxin formation.
3. **Toxic agent**—Toxins produced by *Clostridium botulinum* or *C. parabotulinum* (botulinus bacillus). Most outbreaks are due to toxin from type A or B organisms; a few from type E, mainly related to fish. Toxin is produced in improperly processed food only under anaerobic conditions and particularly in nonacid foods. Toxin is easily destroyed by boiling but spores require higher temperatures.
4. **Reservoir and source of poisoning**—The reservoir of botulinus bacillus is soil and the intestinal tract of animals. Toxin is formed by anaerobic growth of spores in food, which is the immediate source of poisoning.
5. **Mode of transmission**—By ingestion of food usually eaten uncooked, and from jars or cans inadequately processed during canning. Most poisonings in the United States are due to home canned vegetables; pimentos, olives, and mushrooms have been involved in epidemics. In Europe most cases are due to sausages or other smoked or preserved meats.

6. Incubation period—Symptoms usually appear within 18 hours after eating food containing toxin, possibly longer, the interval being determined by amount of contaminated food taken and content of botulinus toxin.

7. Period of communicability—Not applicable.

8. Susceptibility and resistance—Susceptibility is general.

9. Methods of control—

A. Preventive measures:

1. Governmental control by regulation and inspection of commercial processing of canned and preserved foods.
2. Education of housewives and others concerned with home canning of foods in the essentials of safe processing, as to time, pressure, and temperature needed to destroy spores.
3. Education in value of boiling home canned green and leafy vegetables before serving; also thorough cooking of sausage and other meat and fish products.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report obligatory in most states and countries, Class 2A (p. 5). Includes suspect and confirmed cases.
2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None for simple contacts with the patient. Those who have also eaten the suspected food should be given promptly polyvalent botulinus antitoxin or antitoxin of the appropriate type, if available.
7. Investigation of contacts and source of infection: Search for contaminated food. Study of food habits and recent food history of persons attacked.
8. Specific treatment: Intramuscular administration of botulinus antitoxin.

C. Epidemic measures:

1. Suspicion or recognition of a case of botulism should immediately raise the question of a group outbreak. Isolated sporadic cases are uncommon.
2. Immediate search for persons who shared suspected food and for any remaining food from same source that may be similarly contaminated, such food, if found, to be destroyed after samples have been taken for laboratory examination. Search for limberneck in chickens.

D. International measures: None.

BRUCELLOSIS

1. **Identification**—A systemic infection with acute or insidious onset, characterized by continued, intermittent or irregular fever of variable duration, headache, weakness, profuse sweating, chills or chilliness, arthralgia, and generalized aching. The disease may last for several days, many months, or occasionally for several years. Recovery is usual but disa-

bility is often pronounced. The fatality is 2% or less; higher for *B. melitensis* infections than for other varieties. Clinical diagnosis is often difficult and uncertain. Synonyms: Undulant Fever, Malta Fever, Mediterranean Fever.

Laboratory diagnosis is by isolation of the infectious agent from blood, bone marrow or other tissues, or from discharges of the patient. The agglutination test is a valuable aid.

2. **Occurrence**—World-wide, especially Mediterranean countries of Europe, USSR, and North and South America. Males affected more often than females because of occupational hazards. Sporadic cases and outbreaks occur among consumers of unpasteurized milk or milk products from cows and goats. Reported incidence in United States less than 2,000 cases annually, and declining, but reporting is incomplete because of diagnostic difficulties.
3. **Infectious agents**—*Brucella melitensis*; *Brucella abortus*; *Brucella suis*.
4. **Reservoir and source of infection**—Reservoirs of infection are cattle, swine, sheep, goats, and horses. The sources of infection are tissues, blood, urine, milk, and especially placentas, vaginal discharges, and aborted fetuses of infected animals.
5. **Mode of transmission**—By contact with infected animals, animal tissues or secretions, and by ingestion of milk or dairy products from infected animals. Air-borne infection may occur in laboratories.
6. **Incubation period**—Highly variable and difficult to ascertain; onset is insidious and date of infection usually not established; usually 5 to 21 days, occasionally several months.
7. **Period of communicability**—Rarely communicable from man to man; the infectious agent may be discharged in urine and other excretions for long periods.
8. **Susceptibility and resistance**—Susceptibility is variable, as indicated by wide differences in severity and duration of clinical illness, lesser susceptibility of children to manifest disease, and frequency of mild and inapparent infections. Duration of acquired immunity is uncertain.
9. **Methods of control**—Ultimate control of brucellosis in man rests in the elimination of the disease among domestic animals.

A. Preventive measures:

1. Education of farmers and workers in slaughterhouses, packing plants, and butcher shops as to the nature of the disease and the danger of handling carcasses or products of infected animals.
2. Search for infection among livestock by the agglutination reaction and elimination of infected animals from herds by segregation or slaughter. The control of infection among swine usually requires slaughter of the drove.
3. Pasteurization of milk and dairy products from cows, sheep, or goats. Boiling of milk is practicable when pasteurization is not possible.
4. Immunization of calves, and of kids or lambs where milk is used, with vaccines prepared from modified living or killed bacilli.
5. Care in handling and disposal of discharges and fetus from an aborted animal. Disinfection of areas contaminated.
6. Meat inspection and condemning of carcasses of infected swine; not a useful procedure for cattle.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report obligatory in most states and countries, Class 2B (p. 5).

2. Isolation: None.
 3. Concurrent disinfection: Of body discharges.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Trace infection to the common or individual source, usually infected domestic goats, swine, or cattle, or unpasteurized milk or dairy products from cows and goats.
 8. Specific treatment: The tetracycline antibiotics usually produce prompt subsidence of fever and symptoms within several days. The relapse rate is high. More satisfactory results are obtained with a combination of chlortetracycline and dihydrostreptomycin with or without addition of a sulfonamide. Treatment should be continued for at least 3 weeks.
- C. **Epidemic measures:** Search for common vehicle of infection, usually unpasteurized milk or milk products from an infected herd. Stop distribution or provide pasteurization.
- D. **International measures:** Control of domestic animals in international trade and transport.

CANDIDIASIS

1. **Identification**—Candidiasis is a mycosis, most commonly seen in infants and debilitated persons, characterized by production of pseudomembranes on mucosal surfaces, eczematoid skin lesions and rarely granulomata of various tissues. The clinical reaction varies according to location of lesions, age, allergic state of the patient, and other factors. Common manifestations are oral thrush, vaginitis or vulvovaginitis, intertriginous skin lesions of various kinds, bronchopulmonary disease, meningitis, and rarely endocarditis and generalized infection. Skin and local infections are benign; systemic infections have a grave prognosis. Synonyms: Moniliasis, Thrush.

Laboratory confirmation depends upon repeated microscopic demonstration of budding and mycelial forms in direct smears of freshly collected sputum or material from lesions. The fungus multiplies rapidly in collected sputum held at room temperature. It may be found in sputum from patients with tuberculosis, other lung infections, or tumors; it is cultured easily.

2. **Occurrence**—World-wide and sporadic. More common in females than males. Infection of fingers is associated with maceration through long exposure to water (bartenders, housewives). Skin lesions are common in obese persons with excessive perspiration in frictional folds, in diabetics, and in lactating women. Oral and dermal thrush occurs in newborn, premature, and debilitated infants. Oral thrush occurs in older persons with ill-fitting dentures. The fungus is frequently found in patients receiving wide spectrum antibiotic therapy and in patients with cancer; also found in fowl, other animals and fruit.
3. **Infectious agents**—*Candida albicans* (*Monilia albicans*) and occasionally other species of *Candida*. Oral thrush rarely may be caused by *Saccharomyces*.

4. **Reservoir and source of infection**—Reservoir is man; source of infection is secretions of mouth, skin, vagina, and gastrointestinal tract of persons with inapparent infection.
5. **Mode of transmission**—From mother to infant during childbirth, by contact, and by endogenous spread. The frequency of inapparent infections is demonstrated by isolation of *C. albicans* from sputum, oral surfaces or feces of persons ill with other diseases, or from apparently healthy persons. The infrequency with which clinical disease develops through contact with patients suggests that host susceptibility is a more important factor in clinical disease than communicability.
6. **Incubation period**—Variable, 2 to 5 days in thrush of infants.
7. **Period of communicability**—Presumably for the duration of lesions.
8. **Susceptibility and resistance**—Inapparent infections are common. Many adults are hypersensitive to the fungus and possess antibodies. Second attacks are common; clinical manifestations are likely to follow general or locally lowered resistance.
9. **Methods of control**—
 - A. **Preventive measures:** Detection and treatment of vaginal thrush during third trimester of pregnancy.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Official report ordinarily not justifiable, Class 5, p. 6.
 2. Isolation: In nurseries, segregation of patients with oral thrush.
 3. Concurrent disinfection: Of secretions and contaminated articles.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Not profitable in sporadic cases.
 8. Specific treatment: Infections developing during antibiotic therapy sometimes disappear when medication is discontinued. Mycostatin (9) is useful in gastrointestinal infections, amphotericin B in generalized disease. Topical application of sodium caprylate or gentian violet to lesions of skin and mucous membrane.
 - C. **Epidemic measures:** Epidemics are largely limited to thrush in nurseries for the newborn. Cultures should be taken from the mouths of infants during the first 2 days of life. If *Candida albicans* is demonstrated, clinical thrush can be confidently predicted. Such infants and those with oral thrush should be segregated and emphasis on general cleanliness increased. Concurrent and terminal disinfection by cleaning are to be practiced. Compare Epidemic Diarrhea of the Newborn, 9A and 9C (pp. 64, 65).
 - D. **International measures:** None.

CAT SCRATCH FEVER

1. **Identification**—The first manifestation ordinarily is a minor local inflammatory lesion with swelling, erythema and slight ulceration or papule formation at the site of a scratch wound. Subsequent swelling and tenderness of regional lymph nodes proximal to the wound is highly characteristic, with eventual suppuration, the pus being thick, greenish,

and bacteriologically sterile. The swelling gradually recedes without treatment, in most instances within 2 to 3 weeks, but sometimes it persists for several months. Fever is an early symptom, particularly in children, ranging from 37.8° to 40° C (100° – 104° F), variable and sometimes undulating. Chills, rash, malaise, and other manifestations of a generalized infection are of irregular occurrence. Most patients are ambulatory; a chronic form of the disease has been observed. Synonym: Benign Inoculation Lymphoreticulosis.

A skin test with antigen prepared from the pus of lymph nodes is helpful in diagnosis. Complement-fixation with psittacosis-lymphogranuloma venereum group antigen contributes little.

2. **Occurrence**—Apparently world-wide. No seasonal distribution. Affects males and females equally, but young people and children more than others.
3. **Infectious agent**—Unknown. A virus has been isolated from lesions but the suggested relationship to the psittacosis-lymphogranuloma group has not been supported.
4. **Reservoir and source of infection**—The probable reservoir is one of several animals, usually with inapparent infection, most frequently the cat. Saliva and mouth secretions are the probable sources of infection.
5. **Mode of transmission**—The infectious agent is believed to be widely distributed in nature. Animals suggestively become infected and transmit the infection to man by scratching, biting, licking, or other means. Minor trauma after insect bites, or contact with splinters or thorns is advanced as the mode of transmission in the frequent instances of no known direct contact with animals.
6. **Incubation period**—Usually 7 to 14 days from inoculation to primary lesion; possibly as short as 2 days.
7. **Period of communicability**—Unknown for reservoir hosts. Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—Unknown for man; most animals apparently are highly resistant, inapparent infection being usual.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Immediate cleansing and treatment of wounds due to scratches or bites of animals, particularly cats.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
 2. Isolation: None.
 3. Concurrent disinfection: Of discharges from lesion or lymph node.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Examination of family contacts for similar scratch wounds and search for cats or other animal pets as sources of infection.
 8. Specific treatment: None; chloramphenicol, chlortetracycline, and oxytetracycline are stated to alleviate early constitutional symptoms, but not reliably to limit regional lymphadenitis.
 - C. **Epidemic measures:** Not applicable; a sporadic disease.
 - D. **International measures:** None.

CHANCROID

1. **Identification**—An acute localized, self-limiting autoinoculable infectious disease characterized clinically by necrotizing ulcerations at the site of inoculation. Genital lesions frequently are accompanied by painful inflammatory swelling and suppuration of regional lymph nodes. Extragenital lesions of umbilicus, tongue, lip, breast, chin, and bulbar conjunctiva are on record. Synonyms: Ulcus Molle, Chancre Mou, Soft Chancre, and Soft Sore.

Microscopic examination of stained exudate from edges of lesion, bacteriologic culture of pus from buboes, intradermal skin test, autoinoculation, and biopsy are diagnostic aides.

2. **Occurrence**—No particular differences in incidence according to age, race, or sex except as determined by sexual habits. Geographically widespread and in some areas a relatively common disease. Incidence in military forces is sometimes higher than for syphilis.

3. **Infectious agent**—*Hemophilus ducreyi*, Ducrey bacillus.

4. **Reservoir and source of infection**—Reservoir is man. The source of infection is discharges from open lesions and pus from buboes of patients; suggestive evidence that women are occasionally carriers.

5. **Mode of transmission**—Predominantly by direct venereal contact except for rare instances of professionally acquired lesions on hands of physicians and nurses; accidental inoculation of children is known. Indirect transmission is rare. Prostitution, indiscriminate sexual promiscuity, and uncleanness are factors favoring transmission.

6. **Incubation period**—From 3 to 5 days, occasionally longer; if abrasions of mucous membrane are present, as short as 24 hours.

7. **Period of communicability**—As long as the infectious agent persists in the original lesion or discharging regional lymph nodes; usually parallels healing and in most instances a matter of weeks.

8. **Susceptibility and resistance**—Susceptibility is general; no evidence of natural or acquired immunity.

9. **Methods of control**—

A. Preventive measures:

1. Except for specific reference to chancroid as such, and those measures specific for syphilis, the preventive measures are those of syphilis (see 9A, p. 187).

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report obligatory in many states and countries, Class 2B (p. 5).

2. Isolation: None; avoid sexual contact until lesions are healed.

3. Concurrent disinfection: None; ordinary personal cleanliness.

4. Terminal disinfection: None.

5. Quarantine: None.

6. Immunization of contacts: Not applicable; prompt treatment on recognition of the disease or clinical suspicion that it exists.

7. Investigation of contacts and source of infection: Search for sexual contacts of the period 2 weeks before lesions appeared and after the patient developed signs and symptoms.

8. Specific treatment: Sulfonamides (sulfanilamide, sulfathiazole or sulfadiazine); tetracycline antibiotics or chloramphenicol only in case

of sulfonamide resistance because of the possibility of masking syphilis.

C. Epidemic measures: Nondeclining levels of occurrence or any increase in incidence are indication for increased vigilance and more rigid application of measures outlined in 9A and 9B.

D. International measures: (See Syphilis 9D, p. 188).

CHICKENPOX

1. Identification—An acute viral disease of sudden onset with slight fever, mild constitutional symptoms, and an eruption which is maculopapular for a few hours, vesicular for 3 to 4 days, and leaves a granular scab. Lesions tend to be more abundant on covered than on exposed parts of the body; may appear on scalp and mucous membranes of upper respiratory tract; commonly occur in successive crops with several stages of maturity present at the same time; may be so few as to escape observation. Essentially nonfatal; such deaths as occur are almost invariably the result of septic complications or encephalitis. Synonym: Varicella.

Laboratory tests for specifically identifying chickenpox virus, such as tissue culture isolation or various serological means, are useful but not generally available.

2. Occurrence—Nearly universal. In metropolitan communities probably 70% of persons have had the disease by 15 years of age. Not uncommon in early infancy. Winter is the season of greatest prevalence in temperate zones.

3. Infectious agent—The virus of chickenpox. The occasional development of chickenpox in persons exposed to herpes zoster has suggested that the two diseases are different manifestations of the same viral infection. This concept is supported by the observed similarity of morphology of the 2 agents in tissue culture, and an antigenic agreement based on complement-fixation and neutralization tests.

4. Reservoir and source of infection—Reservoir is the infected person. Source of infection is secretions of the respiratory tract; lesions of the skin are of little consequence and scabs of themselves are not infective.

5. Mode of transmission—From person to person by direct contact, droplet, or air-borne spread; indirectly through articles freshly soiled by discharges from the skin and mucous membranes of infected persons. One of the most readily communicable of diseases, especially in the early stages of the eruption.

6. Incubation period—From 2 to 3 weeks; commonly 13 to 17 days.

7. Period of communicability—Probably not more than 1 day before nor more than 6 days after the appearance of the first crop of vesicles.

8. Susceptibility and resistance—Susceptibility is universal among those not previously attacked; ordinarily a more severe disease of adults than of children. An attack confers long immunity; second attacks are rare.

9. Methods of control—

A. Preventive measures:

1. The chief public health importance of this disease is that cases thought to be chickenpox in persons over 15 years of age or of any age during an epidemic of smallpox, should be viewed with suspicion and investigated to eliminate possibility of smallpox.

5596
3/10/62

2. Several investigators have noted fatal chickenpox in patients receiving steroid therapy. If nonimmune, effort should be made to protect such persons against exposure to chickenpox.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Official report is not ordinarily justifiable, Class 5 (p. 6). Case report of chickenpox in adults (Class 3B) may be required where smallpox is infrequent.
2. Isolation: None; exclusion from school for 1 week after eruption first appears and avoid contact with susceptibles.
3. Concurrent disinfection: Articles soiled by discharges from the nose and throat and from lesions.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Of no practical importance, except under suspicion of smallpox.
8. Specific treatment: None.

C. Epidemic measures: None.

D. International measures: None.

CHOLERA

1. **Identification**—A serious acute intestinal infection characterized by sudden onset, vomiting, profuse watery diarrhea, rapid dehydration, and collapse. Severity differs greatly from place to place and within epidemics; mild cases show only diarrhea. Death may occur within a few hours of onset. Epidemics tend to be explosive, and fatality varies from 5 to 75%.

Cholera vibrios can be cultivated from feces by appropriate bacteriologic means.

2. **Occurrence**—Endemic in parts of India and East Pakistan. From these centers, spreads occasionally along lines of communication to nearby countries, sometimes causing extensive epidemics. Absent in epidemic proportions from Europe and the Western Hemisphere for many years although it has invaded repeatedly.
3. **Infectious agent**—*Vibrio cholerae*, cholera vibrio.
4. **Reservoir and source of infection**—Reservoir is an infected person; source of infection is feces and vomitus of patients, to a lesser extent feces of persons incubating the disease and of convalescents.
5. **Mode of transmission**—Transmission in the initial wave of an epidemic of cholera is regularly by contaminated water. Subsequent cases ordinarily occur through passage of the vibrio by foods contaminated by soiled hands or utensils and by flies. Water has a less important part in sporadic and endemic cholera; transmission is mainly by food, hand to mouth transfer of contaminated material, and by flies. Carriers are not a significant factor in spread of the infection.
6. **Incubation period**—From a few hours to 5 days, usually 3 days.
7. **Period of communicability**—While cholera vibrios are present in feces and vomitus; for feces, usually 7 to 14 days after onset.

8. Susceptibility and resistance—Susceptibility is general, although variable. Recovery from clinical attack appears to afford some protection for several years. Immunity artificially induced by vaccines is of variable degree and uncertain duration, but not more than 6 months.

9. Methods of control—

A. Preventive measures:

1. Sanitary disposal of human feces.
2. Protection and purification of water supplies, see Typhoid Fever 9A1 (p. 209).
3. Boiling of milk or pasteurization of milk and dairy products, see Typhoid Fever 9A3 (pp. 209, 210).
4. Sanitary supervision of processing, preparation, and serving of foods, especially those eaten moist and raw; special attention to provision and use of hand-washing facilities.
5. Fly control, control of fly breeding, and screening to protect foods against fly contamination, see Typhoid Fever 9A6 (p. 210).
6. Education of the public in habits of personal cleanliness, especially washing the hands before eating and after defecation.
7. Active immunization with cholera vaccine of persons subject to unusual or continued risk.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report universally required by international regulations, Class 1 (p. 5).
2. Isolation: In hospital or screened room during communicable period.
3. Concurrent disinfection: Of feces and vomitus, and of articles used by patient. Practice by attendants of scrupulous cleanliness; disinfection of hands each time after handling or touching articles contaminated by feces.
4. Terminal disinfection: Thorough cleaning, including premises occupied by patient if removed to hospital.
5. Quarantine: Surveillance of contacts for 5 days from last exposure and longer if feces contain cholera vibrios.
6. Immunization of contacts: No passive immunization. Inoculation of contacts with cholera vaccine can be expected to do no more than protect against subsequent or continued exposure. (See C3 *infra*.)
7. Investigation of contacts and source of infection: Search for unreported cases. Investigate possibilities of infection from polluted drinking water or from contaminated uncooked foods.
8. Specific treatment: None. Prompt and adequate replacement of fluid and electrolyte and other measures for shock are paramount.

C. Epidemic measures:

1. Emergency measures to assure adequate water supply; boiling of water used for drinking, toilet purposes, or washing dishes or food containers, unless water supply is treated adequately as by chlorination and protected from contamination thereafter.
2. Inspection service for early detection of infected persons; provision of temporary emergency facilities by requisition or otherwise, for isolation of patients and suspects. Identification and isolation of carriers desirable but usually impracticable. Detention in suitable camps for 7 days, of those desirous of leaving for another locality.
3. Immediate administration of cholera vaccine to exposed population groups, despite its limited value.

4. Careful supervision of food and drink. After cooking or boiling should be protected against contamination, as by flies and human handling.
5. Control of flies, by limiting fly breeding, by use of appropriate insecticides, and by screening kitchens and eating places. See Typhoid Fever 9A6 (p. 210).
6. Promulgation of temporary regulations to assure proper execution of the above control measures.

D. International measures:

1. Telegraphic notification of WHO and adjacent countries, by governments, of a first case of cholera of local origin.
2. Measures applicable to ships, aircraft, and land transport arriving from cholera areas are specified in International Sanitary Regulations, World Health Organization, annotated edition, Geneva, 1957.
3. International travelers: Cholera patients or suspects are not permitted to depart for another country. Many countries require travelers from an endemic cholera area to possess a valid international cholera vaccination certificate for entry. The certificate is valid from 6 days until 6 months after the first injection of vaccine, or in the event of revaccination within such period of 6 months, then as of that date and for 6 months thereafter.

CHROMOBLASTOMYCOSIS

1. **Identification**—A chronic spreading but usually localized mycosis of the skin and subcutaneous tissues, usually of a lower extremity. Hematogenous spread to the brain has been reported. Progression of the lesion to contiguous tissues is slow, over a period of years, with eventual large verrucous or even cauliflower-like masses, and lymph stasis; of itself rarely a cause of death. Synonyms: Chromomycosis, Dermatitis Verrucosa.

Microscopic examination of scrapings from lesions reveals characteristic brown fungus cells (hyphae in superficial crusts, single or multiple sclerotoid cells in cutaneous and subcutaneous tissues) which can be cultured.

2. **Occurrence**—World-wide distribution as sporadic cases from widely scattered areas but mainly Central America, South America, Australia, and the West Indies. Primarily a disease of rural tropical regions, probably because of more frequent penetrating wounds of feet not protected by shoes. The infection is commonest in men aged 30 to 50 years; females are rarely infected.
3. **Infectious agents**—*Phialophora verrucosa*, *P. pedrosoi* (Hormodendrum, Fonsecaea), *P. compactum*, and species of *Cladosporium* (Hormodendrum).
4. **Reservoir and source of infection**—Reservoir presumably is wood, soil, or vegetation; source of infection the same.
5. **Mode of transmission**—Traumatic contact with contaminated wood or other materials.
6. **Incubation period**—Unknown; probably weeks or months.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—Unknown, but rarity of disease and absence of laboratory infections suggest man is relatively resistant.

9. Methods of control—

A. Preventive measures: None.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
2. Isolation: None.
3. Concurrent disinfection: Of discharges from lesions and articles soiled therewith.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Not applicable.
7. Investigation of contacts and source of infection: Not profitable.
8. Specific treatment: None; iodides, copper sulfate and sulfur should be tried. Stilbamidine or 2-hydroxystilbamidine may be used with care systemically, and gentian violet and propamidine locally. Some success has been reported with calciferol.

C. Epidemic measures: Not applicable, a sporadic disease.

D. International measures: None.

CLONORCHIASIS

1. Identification—An infection of the bile ducts with the Asiatic liver fluke of man. Clinical reaction may be slight or absent in light infections. Symptoms result from local irritation of bile ducts by flukes, from systemic toxemia, and possibly from secondary bacterial invaders. Loss of appetite, diarrhea, and sensations of abdominal pressure are common early symptoms. Bile duct obstruction, rarely producing jaundice, may be followed by cirrhosis, enlargement and tenderness of the liver, and progressive ascites and edema. Eosinophilia is frequent, 5 to 40%. A long continued disease, sometimes 20 years or more, but uncommonly either a direct or contributing cause of death. Synonym: Chinese Liver Fluke Disease.

Direct diagnosis depends on finding the characteristic fluke eggs in feces or by duodenal drainage. Differentiation is required from eggs of heterophyid and opisthorchid flukes, producing disease in cats and dogs but also heterophyidiasis and opisthorchiasis in man in Southeast Asia (Thailand), Egypt, Russia, and other countries.

2. Occurrence—Highly endemic in Southeast China, present throughout the country except northwest; Japan, widespread; Taiwan; South Korea and Vietnam, principally Red River Delta. Imported cases in immigrants in other parts of the world. Native Hawaiians contract disease from imported fish. Extensive epidemics have occurred among refugees from endemic areas and in other unusual circumstances.

3. Infectious agent—*Clonorchis sinensis*.

4. Reservoir and source of infection—Man, cat, dog, hog, and other animals are reservoir hosts of adult flukes. Source of infection is fresh-water fish eaten raw (fresh, dried, salted, or pickled) or partly cooked.

5. Mode of transmission—Eggs deposited in the bile passages are evacuated in feces. Eggs in feces contain a fully developed miracidium; if ingested by a susceptible snail (*Buliminae* of the family *Amnicolidae*), hatches in its intestine and penetrates vascular spaces. Cercariae develop and

emerge into water; on contact with second intermediate host, fresh-water fishes (*Cyprinidae*) of which 40 or more species are appropriate, cercariae penetrate the host and encyst, usually in muscle, occasionally on underside of scales. Man or other definitive host is infected by eating fish; during digestion, larvae are set free from cysts, and migrate via the common bile duct to biliary capillaries. The complete life cycle, from man to man, requires at least 3 months.

6. **Incubation period**—Undetermined; flukes reach maturity within 16 to 25 days after encysted larvae are ingested.
7. **Period of communicability**—Infected individuals may pass viable eggs for as long as 15 to 20 years. Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—Susceptibility is universal. No resistance develops with age; in endemic areas, highest incidence at age 55 to 60 years.
9. **Methods of control**—

A. Preventive measures:

1. In endemic areas, education of the public regarding life cycle of parasite.
2. Thorough cooking of all fresh-water fish.
3. Treatment or storage of night soil before use as fertilizer in fish ponds: 1 part of 0.7% solution of ammonium sulfate to 10 parts of feces will kill eggs in 30 minutes.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3C (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: Sanitary disposal of feces.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Not applicable.
7. Investigation of contacts and source of infection: Of the individual case, unprofitable. A community problem (see 9C below).
8. Specific treatment: None; chloroquine hydrochloride and gentian violet medicinal are of some value.

C. Epidemic measures: Locate source of infected fish parasitized through use of night soil as fertilizer in fish ponds, or through fecal contamination of streams. Shipments of dried or pickled fish are the likely source in nonendemic areas.

D. International measures: Control of fish products imported from endemic areas.

COCCIDIOIDOMYCOSIS

1. **Identification**—A systemic mycosis beginning as a respiratory infection and following one of two courses.

A. Primary infection: May be entirely asymptomatic or resemble an acute febrile influenza-like illness with fever, chills, cough, and pleural pain. About one-fifth of clinically recognized cases (an estimated 8% of all primary infections), develop erythema nodosum (Vallbo, Feyer), or

more rarely, erythema multiforme, a complication most frequent in white females and rarest in Negro males. Primary infection may (1) heal completely without detectable residuals, (2) leave radiographic fibrosis or calcification of pulmonary lesions, (3) leave a persistent thin-walled cavity, or (4) and most rarely, progress to the disseminated form of the disease comparable to progressive primary tuberculosis.

The fungus may be found in sputum by direct examination or by culture. Reaction to skin test with coccidioidin appears within 2 days to 3 weeks after onset; precipitin and complement-fixation tests are usually positive at an early date.

B. Coccidioidal granuloma: A progressive highly fatal granulomatous disease characterized by lung lesions and single or aggregated abscesses throughout the body, especially in subcutaneous tissues, skin, bone, peritoneum, testes, thyroid, and central nervous system. Coccidioidal meningitis resembles tuberculous meningitis.

The fungus can be demonstrated in sputum and in materials from lesions by microscopic examination and by culture.

A granulomatous disease, sometimes referred to as paracoccidioidal granuloma, has no relation to coccidioidomycosis (see South American Blastomycosis, p. 43).

2. **Occurrence**—Primary infections are extremely common in scattered highly endemic arid and semiarid areas; in United States from Southern California to west Texas; in northern Argentina, Mexico, and Russia, in areas having similar climate and terrain. Affects all ages, both sexes, and all races. Infection most frequent in summer, especially after wind and dust storms. Coccidioidal granuloma has the geographic distribution of Valley Fever. More than half of cases occur between 15 and 25 years of age; males are infected 5 times more frequently than females; and dark-skinned individuals 10 times more often than others. An important disease among military recruits who train in endemic areas.
3. **Infectious agent**—*Coccidioides immitis*, a dimorphic fungus growing in soil and in culture media as a mold which reproduces by arthrospores, and in tissue and under special conditions of culture as spherical cells which become sporangia and produce sporangiospores (endospores).
4. **Reservoir and source of infection**—Reservoir is soil; fungus apparently propagates in soil, especially in and around rodent burrows, in arid and semiarid regions where temperature and moisture requirements are satisfactory, to infect man, cattle, dogs, horses, burros, sheep, swine, and wild rodents. Source of infection is soil and spore-laden dust.
5. **Mode of transmission**—Inhalation of spores in dust from soil and dry vegetation, and in laboratories inhalation of spores from cultures. Infection through open wounds is possible but infrequent. Draining lesions and expectorated sputum may in rare instances contaminate articles and conceivably permit indirect transmission following conversion of the fungus from parasitic to saprophytic phase.
6. **Incubation period**—10 days to 3 weeks in primary infection. Coccidioidal granuloma develops insidiously, not necessarily preceded by recognized symptoms of primary pulmonary infection.
7. **Period of communicability**—Under natural conditions not directly transmissible from animal or man to man.
8. **Susceptibility and resistance**—Susceptibility to primary infection is general as indicated by high incidence of positive coccidioidin reactors in endemic areas; recovery apparently is followed by solid immunity.

9. Methods of control—

A. Preventive measures:

1. In endemic areas, planting of grass, oiling of air fields and other dust control measures. Individuals from nonendemic areas should not be recruited to dusty occupations, such as road building and picking cotton.
2. Destruction of diseased dogs and rodents to avoid contamination of the environment.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report of recognized coccidioidal disease in selected endemic areas (USA); in many countries not a reportable disease, Class 3B (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: Of discharges and soiled articles.
4. Terminal disinfection: Thorough cleaning.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Not profitable except in cases appearing in nonendemic areas where residence and travel history should be obtained.
8. Specific treatment: None. Amphotericin B appears of some benefit in disseminated infections but must be given near the toxic level.

C. *Epidemic measures*: Epidemics occur only when groups of susceptibles are infected by air-borne spores. Dust control measures should be instituted where practicable.

D. *International measures*: None.

COLORADO TICK FEVER, VIRAL

1. **Identification**—An acute febrile disease with an erythematous rash; a brief remission is usual, followed by a second bout of fever, each of 2 or 3 days' duration. The disease is characteristically mild but may be severe in children, occasionally with a resulting encephalitis or tendency to bleed; deaths are uncommon. One of the group of Arthropod-Borne Viral Fevers, (p. 33).

Laboratory confirmation is preferably by isolation of virus; complement fixing and neutralizing antibodies do not appear until 2 or more weeks after onset.

2. **Occurrence**—Only known area of occurrence is the United States; and present there in the states of Washington, Oregon, California, Nevada, Utah, Idaho, Montana, Wyoming, Colorado, and South Dakota. Virus has been isolated from *D. andersoni* in British Columbia and in New Mexico. The disease is most frequent in adult males, but commonly affects children and women; seasonal incidence corresponds to greatest tick activity; endemic distribution, and a common disease in much of the affected area.

3. **Infectious agent**—The virus of Colorado tick fever.

4. **Reservoir and source of infection**—Reservoir is small mammals, of which ground squirrels and the porcupine have been identified, together with nymphal and larval ticks. Source of infection is the adult vector tick.

5. **Mode of transmission**—Immature ticks, *Dermacentor andersoni*, acquire infection through feeding on infected animals during viremia; they remain infected through the various moults and transmit virus to man by feeding as adult ticks.
6. **Incubation period**—Usually 4 to 5 days.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man; the wild life cycle maintained by larval and nymphal ticks is the important consideration. Ticks remain infective throughout life. Virus is present in man during febrile course, from 1 to 10 days after onset.
8. **Susceptibility and resistance**—Susceptibility apparently universal. Second attacks are unknown; experimental reinfection unsuccessful.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Control of ticks; see Rocky Mountain Spotted Fever, 9A1 and 9A2 (p. 160).
 2. No available vaccine.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: In endemic areas (USA); in most states and countries not a reportable disease, Class 3B (pp. 5, 6).
 2. Isolation: None.
 3. Concurrent disinfection: None; ticks on patient should be destroyed.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Identification of ticks and tick infested areas.
 8. Specific treatment: None.
 - C. **Epidemic measures:** Not applicable.
 - D. **International measures:** None.

CONJUNCTIVITIS, ACUTE BACTERIAL

1. **Identification**—The disease begins with lacrimation, irritation, and vascular injection of the palpebral and bulbar conjunctivae of one or both eyes, followed by edema of the lids, photophobia, and a mucopurulent exudate; in severe cases, ecchymoses of the bulbar conjunctiva and marginal infiltration in the cornea. A nonfatal disease with usual clinical course of 2 to 3 weeks; many patients have no more than vascular injection of the conjunctivae and slight exudate for a few days. Synonyms: Sore Eyes, Pink Eye.

Confirmation of clinical diagnosis is by bacteriologic culture, or microscopic examination of smears of exudate.

Other forms of acute conjunctivitis are to be differentiated: Acute conjunctivitis of the newborn to include gonorrheal ophthalmia and ophthalmia neonatorum (p. 86), trachoma (p. 194), epidemic keratoconjunctivitis (p. 97), pharyngoconjunctival fever (p. 215) and inclusion blennorrhoea.

2. **Occurrence**—Widespread throughout the world, particularly in warmer climates; frequently epidemic. In the United States, infection with *H. aegyptius* is largely confined to rural areas of southernmost states, Georgia to California, primarily during summer and early autumn in those areas an important cause of absence from school. Occurs throughout the United States, often in association with acute respiratory infection during cold seasons.
3. **Infectious agents**—*Haemophilus aegyptius* (Koch-Weeks bacillus) appears to be the most important; *Haemophilus influenzae*, *Moraxella lacunata*, staphylococci, streptococci, pneumococci, and *C. diphtheriae* may produce the disease.
4. **Reservoir and source of infection**—Reservoir is man. Source of infection is discharges from the conjunctiva or upper respiratory tract of infected persons, possibly including carriers.
5. **Mode of transmission**—Contact with infected individuals through contaminated fingers, clothing, or other articles. In some areas may be mechanically transmitted by eye-gnats or flies, but importance as a vector undetermined and probably differs from area to area.
6. **Incubation period**—Usually 24 to 72 hours.
7. **Period of communicability**—During the course of active infection.
8. **Susceptibility and resistance**—Children under 5 years are most often affected and incidence decreases with age. The debilitated and aged are particularly susceptible to staphylococcal infections. Immunity after attack is low-grade and variable, according to the infectious agent involved.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Personal cleanliness, hygienic care and treatment of affected eyes.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Obligatory report of epidemics; no case report, Class 4 (p. 6).
 2. Isolation: None. Children should not attend school during the acute stage.
 3. Concurrent disinfection: Of discharges and soiled articles.
 4. Terminal disinfection: Thorough cleaning.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Usually not profitable.
 8. Specific treatment: Local application of a tetracycline antibiotic or a sulfonamide such as sodium sulfacetamide.
 - C. **Epidemic measures:**
 1. Adequate and intensive treatment of patients and their associates.
 2. In areas where insects are suspected of mechanically transmitting infection, measures to prevent access of eye-gnats or flies to eyes of sick and well persons.
 3. Insect control, according to the suspected vector.
 - D. **International measures:** None.

CRYPTOCOCCOSIS

1. **Identification**—A mycosis most frequently recognized in the United States as a chronic, usually fatal meningitis, cryptococcal meningitis (*Torula meningitis*); pulmonary lesions precede meningeal involvement and may heal without further progression. The fungus also produces acne-like skin lesions, subcutaneous tumor-like masses, acute pneumonitis, and occasionally a generalized infection involving diverse organs. Synonyms: *Torulosis*, European *Blastomycosis*.

Cryptococcal meningitis is confirmed by microscopic examination of spinal fluid or pus. The fungus should be cultured, with ability to grow at 37° C. and pathogenicity for mice determined. Serologic tests not reliable. Some serums show significant titers to *Histoplasma* antigens.
2. **Occurrence**—Sporadic cases occur in all parts of the world. All races are susceptible; males infected twice as frequently as females; all ages affected but mainly between 20 and 40 years. An occasional cause of epidemic intractable mastitis in cattle; occurs also in cats, dogs, horses, monkeys, and other animals.
3. **Infectious agent**—*Cryptococcus neoformans* (*Torula histolytica*).
4. **Reservoir and source of infection**—Reservoir is saprophytic growth in external environment. Can be isolated consistently in many parts of the world from old pigeon nests and pigeon droppings under roosting locations. Source of infection is spore-laden dust.
5. **Mode of transmission**—Presumably by inhalation of spore-laden dust.
6. **Incubation period**—Unknown. Roentgenographic examination and biopsy have demonstrated inapparent pulmonary infection prior to or without meningitis.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—The frequency of *Cryptococcus* in certain environments, the sporadic occurrence of meningitis and only occasional demonstration of the fungus in normal persons suggest that man has an appreciable resistance.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Destruction of diseased animals. Elimination of pigeons.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Official report ordinarily not justifiable, Class 5 (p. 6).
 2. Isolation: None.
 3. Concurrent disinfection: Of discharges and contaminated dressings.
 4. Terminal disinfection: Thorough cleaning.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Unprofitable in the usual sporadic case. Investigate common exposure to accumulations of pigeon manure in familial or group cases of acute pneumonitis.
 8. Specific treatment: Amphotericin B appears to be effective in some cases.
 - C. **Epidemic measures:** Although currently recognized only as a sporadic disease, epidemics of acute pneumonitis are postulated under conditions of common exposure to known sources of infection, as in clean-

ing or dismantling buildings long occupied by pigeons. *C. neoformans* is almost constantly present in such environments. Protection by masks of persons so exposed is advised.

D. International measures: None.

DENGUE

1. **Identification**—An acute febrile infection of sharp onset, occasionally with 2 paroxysms of short duration; fever for about 5 days and rarely more than 7, intense headache, joint and muscle pains, and eruption. Eruption usually appears 3 to 4 days after onset of fever, either maculopapular or scarlatiniform; petechiae may appear on feet, legs, axillae, or palate on last day of fever or shortly thereafter. Leucopenia is usual, with an absolute decrease of segmented neutrophils and a marked increase of nonsegmented forms. Epidemics are explosive and fatality is exceedingly low. One of the group of Arthropod-Borne Viral Fevers, p. 33. Synonym: Breakbone Fever.

Laboratory tests contributing to identification include hemagglutination, complement-fixation or neutralization techniques, using specific type of virus.

2. **Occurrence**—Endemic areas are limited to parts of the world where mosquito vectors survive in large numbers throughout the year; also may depend on continued immigration of susceptibles such as occurs during war and on distant military posts. Islands of the Southwest Pacific, Vietnam, Laos, Cambodia, Indonesia, India, and northern Australia are areas commonly involved. Epidemics can occur wherever the vectors are present.
3. **Infectious agent**—The viruses of dengue fever; at least 2 immunologically distinct types have been identified in dengue fever. Two other immunologic types are responsible for a different disease (see Arthropod-Borne Viral Hemorrhagic Fevers, p. 34).
4. **Reservoir and source of infection**—Man together with the mosquito is one reservoir; the existence of an added animal-mosquito reservoir is suspected but not demonstrated. The immediate source of infection is an infective vector mosquito.
5. **Mode of transmission**—By the bite of mosquitoes, *Aedes aegypti*, *Aedes albopictus*, or one of the *Aedes scutellaris* complex, infected by biting a patient.
6. **Incubation period**—Three to 15 days, commonly 5 or 6 days.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Patients are usually infective for mosquitoes from the day before onset to the fifth day of disease. The mosquito becomes infective from 8 to 11 days after the blood meal and remains so for life.
8. **Susceptibility and resistance**—Susceptibility is apparently universal. Homologous immunity to either type of virus is of long duration; heterologous immunity, though present, is brief and may permit mild febrile illness without rash.

9. Methods of control—

A. Preventive measures:

1. Measures directed toward elimination of vector mosquitoes, and where practicable their breeding places.

2. Screening of rooms.
 3. Use of mosquito repellents. (See Malaria 9A4, p. 112).
- B. Control of patient, contacts, and the immediate environment:**
1. Report to local health authority: Obligatory report of epidemics; no case report, Class 4 (p. 6).
 2. Isolation: Patient should be kept in screened room for at least 5 days after onset, or in quarters treated with insecticide with residual effect, such as DDT.
 3. Concurrent disinfection: None.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Place of residence of patient during fortnight previous to onset. Search for unreported or undiagnosed cases.
 8. Specific treatment: None.
- C. Epidemic measures:**
1. Community survey to determine density of vector mosquitoes, to identify their breeding places, and to promote plans for their elimination.
 2. Search for and destruction of *Aedes* mosquitoes in places of human habitation.
 3. Use of mosquito repellents by persons exposed through occupation or necessity to bites of vector mosquitoes.
- D. International measures:**
1. Enforcement of provisions of international agreements designed to prevent spread of the disease by persons, and mosquito transfer by ships, airplanes, and land transport from areas where an epidemic exists to areas free of the disease.
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DIARRHEA OF THE NEWBORN, EPIDEMIC

A variety of diseases of children aged less than 2 years, having diarrhea and usually fever as the common clinical manifestation is grouped under the general term of infantile enteritis. Two broad classes are recognized. The larger and generally more serious group includes specific infections that result in primary infectious enteritis. Some are due to known pathogenic agents such as *Salmonella* (p. 162) and *Shigella* (p. 71); some are associated with serologically recognizable types of *Escherichia coli* or a number of other common bacteria; and others may be caused by filterable viruses. The second class includes various forms of secondary enteritis associated with parenteral infection. The several kinds of primary enteritis cannot be distinguished accurately by clinical methods. A number are seen principally among older infants; any one may attack the newborn.

The condition described here as epidemic diarrhea of the newborn is a form of acute enteritis of young infants with characteristics sufficiently distinctive to be recognized individually as an epidemiologic entity by reason of its practical limitation to outbreaks among infants housed in hospital nurseries, and as a clinical entity by manifestations which set it apart from the usual infantile diarrhea. It is not an etiologic entity.

1. **Identification**—An acute communicable disease characterized by severe diarrhea with watery feces containing little or no mucus and no blood, by dehydration, and commonly acidosis. Signs other than of enteric infection are lacking in uncomplicated cases; the temperature is normal or only slightly elevated except with severe dehydration or with pneumonia or other complication. The disorder spreads rapidly from infant to infant in a nursery for the newborn and ordinarily has a high fatality, varying, however, from zero to 40%.

Stool culture and serologic study serve to identify specific infections with known pathogenic bacteria or viruses. Post mortem examination shows remarkably few changes and none pathognomonic of the disorder.

2. **Occurrence**—Frequent in North America and Europe, and probably more widespread than reports from other countries indicate. Primarily a disease of hospitals caring for newborn infants in nurseries. No definite seasonal incidence.
3. **Infectious agents**—Probably more than one infectious agent is responsible. Some outbreaks have been attributed to viruses; others are associated with the presence in the intestine of pathogenic serotypes of *E. coli* of which many are now recognized.
4. **Reservoir and source of infection**—Reservoir is unknown; presumably infected persons. The specific types of *E. coli* appear to be present only among patients and close contacts. Nurses, physicians, and other adults sometimes serve as healthy carriers. Source of infection is secretions or excretions of infected persons or articles contaminated therewith.
5. **Mode of transmission**—The principal mode is probably direct fecal contamination because often the organism is present in nearly pure culture. This relates to poor personal toilet hygiene on the part of the staff, failure to wash hands following each diaper change, and before feeding of infants. Additional faults of aseptic technic include bathing of infants at a common table; weighing at common scales without using sterile pads for each infant; preparation, transportation, and refrigeration of formulas; changing from bottle cap to nipple; and saving unused portions of formula for later feedings. In epidemics associated with specific types of *E. coli*, the whole of the patient's environment is contaminated and the organism can be demonstrated on bed clothing and in dust of the ward.
6. **Incubation period**—Unknown; estimates are from 1 to 21 days, most frequently 2 to 4 days.
7. **Period of communicability**—Readily communicable among newborn infants as long as symptoms are present or a carrier state persists.
8. **Susceptibility and resistance**—Restricted in general to infants under 1 month of age, most frequently those from 8 to 9 days old. Premature infants are more susceptible and have a decidedly higher fatality. Older infants and adults seem to be less susceptible; most other bacterial and viral diseases producing diarrhea in the newborn also affect adults. No known means to induce immunity artificially.
9. **Methods of control**—Current practice is empirical, with principal dependence on general measures for limiting spread of infection and for conduct of a clean nursery.

A. Preventive measures:

1. A nursery for the newborn or premature preferably should accommodate no more than 12 infants; should not communicate directly with other nurseries; have at least 24 square feet of floor space per infant; and provide mechanically controlled running hot and cold water for hand washing. A "suspect" nursery should be provided

to which infants can be transferred on the slightest suspicion of illness. Infants with established illness should be transferred to a separate isolation nursery or to the pediatric service. Because of the ease with which the infection spreads, once introduced into a nursery, the increasing practice of keeping each baby with its mother has much to recommend it.

2. Individual equipment kept at the bassinet should be provided each infant; no common bathing or dressing tables and no bassinet stands for holding or transporting more than 1 infant at a time (see 5, above).
3. No nurse to care for more than 12 infants and their equipment.
4. Feeding formulas, including glucose-water, should be prepared aseptically, placed in clean bottles with clean nipples attached and covered with a cap. The entire product should be subjected to terminal heating, either by steam under pressure, 15 lbs at 121.1° C (250° F) for 5 minutes, or 6 lbs at 110° C (230° F) for 10 minutes, or by flowing steam, 100° C (212° F) for 30 minutes; then refrigerated with nipple caps left on bottles until feeding time. Periodic bacteriological sampling of heated formulas is recommended; coliform organisms should be absent and the total plate count should not exceed 10 bacteria per ml.
5. Normal newborn infants should not be kept in the same nursery with sick infants or older children. An infant born outside the hospital or to a mother who has diarrheal or respiratory illness should not be admitted to the nursery for well infants except after quarantine for at least 6 days, preferably with stool examination for potential pathogens. Nurses caring for patients should have no association with nurseries for normal newborn or premature infants; nurses engaged in the milk kitchen should not attend the infants' toilet; control of visitors to minimize spread of infection; laundry procedures to assure absence of pathogenic organisms from finished product as returned to nursery.
6. Systematic daily record of number and consistency of stools for each infant.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6). Two or more concurrent cases in a nursery are to be interpreted as an epidemic.
2. Isolation: Of infected infant, also suspects.
3. Concurrent disinfection: Of all discharges and articles soiled therewith.
4. Terminal disinfection: Thorough cleaning of nursery and equipment.
5. Quarantine: Complete quarantine of all newborn contacts.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: See 9C2.
8. Specific treatment: The tetracycline antibiotics, chloramphenicol, neomycin, and sulfadiazine have been used in illnesses due to coliform group.

C. Epidemic measures:

1. Permit no new admissions to the contaminated hospital nursery; suspend maternity service unless uninfected nurseries with separate personnel and facilities are available. Exposed babies in the con-

taminated nursery should be cared for by separate medical and nursing personnel skilled in care of communicable disease; observe contacts for at least 2 weeks after last case leaves the nursery; promptly remove each new case to isolation. Maternity service may be resumed after discharge of all contact babies and mothers and thorough cleaning. Put into practice recommendations of 9A so far as feasible in the emergency.

2. Epidemiologic investigation: (a) Assure adequate treatment of missed cases by follow-up examination of all infants discharged from hospital during 2 weeks preceding first recognized case; (b) examine mothers and maternity service personnel for early signs of illness; (c) bacteriologic examination of feces of all sick and exposed babies, mothers and maternity service personnel to detect missed cases and carriers due to a known pathogen; (d) survey hospital for sanitary hazards; (e) investigate preparation of feeding formulas for adequacy of sterilization and refrigeration; bacteriologic examination of solutions and sugars used in formulas and their storage; bacteriologic examination of rubber nipples and bottle caps; (f) inquiry into technique of aseptic nursing of infants, of changing diapers, and of laundering diapers and other clothing.

D. International measures: None.

DIPHTHERIA

1. **Identification**—An acute febrile infection, generally of tonsils, throat, and nose, marked by a patch or patches of grayish membrane. Occasionally, and especially in adults, there is only slight inflammation with little or no membrane. Inapparent infections of the upper respiratory tract outnumber recognized cases. Nasal diphtheria is commonly marked by one-sided nasal discharge and excoriated nares. Nonrespiratory forms include infection of skin and wound surfaces; rarely the vagina. Cutaneous diphtheria usually appears as localized punched-out ulcers. Late effects of absorption of toxin include cranial nerve palsies and myocarditis. Fatality is variable; in some epidemics 10 to 12%, and commonly 2 to 5%.

Diagnosis is by clinical symptoms with confirmation by bacteriologic examination of discharges. Failure to demonstrate the bacillus in suspected diphtheria is not a valid reason for withholding specific treatment. Physicians should continually suspect the possibility of diphtheria in the differential diagnosis of streptococcal or staphylococcal pharyngitis, Vincent infection, infectious mononucleosis, syphilis, and candidal infections, especially following antibiotics. The tendency to prescribe antibiotics automatically for all sore throats on the assumption that most are streptococcal in origin, delays definitive diagnosis and therapy, and is responsible for many fatal cases of diphtheria.

2. **Occurrence**—Endemic and epidemic; a disease of autumn and winter months. In communities where active immunization has been neglected, approximately one-fourth of cases and one half of deaths occur in children under 5 years of age. In communities where childhood immunization has been adequate but reinforcing doses of toxoid were not continued, age distribution tends toward older persons. Clinical disease is more common in temperate zones than in the tropics, although infection rates are often much the same. Relatively, the tropics have more in-

apparent infection, less faucial diphtheria and more diphtheria of the skin.

3. **Infectious agent**—*Corynebacterium diphtheriae*, the Klebs-Loeffler bacillus.
4. **Reservoir and source of infection**—Reservoir is the infected person; sources of infection are discharges and secretions from mucous surfaces of nose, pharynx and nasopharynx, and from skin and other lesions.
5. **Mode of transmission**—Contact with a patient or carrier or with articles soiled with discharges of such persons. Milk has served as a vehicle.
6. **Incubation period**—Usually 2 to 5 days, occasionally longer.
7. **Period of communicability**—Variable, until virulent bacilli have disappeared from secretions and lesions; usually 2 weeks or less, seldom more than 4 weeks.
8. **Susceptibility and resistance**—Infants born of immune mothers are relatively immune, a passive protection usually lost before the sixth month; in North America, probably not more than half of mothers are immune. Recovery from an attack of the disease is usually but not always followed by persisting immunity. Immunity is often acquired through unrecognized infection. Passive temporary immunity of from 10 days to 3 weeks and active immunity of prolonged duration can be induced artificially.
9. **Methods of control**—

A. Preventive measures:

1. The only effectual control of diphtheria is through active immunization on a population basis. All children should be inoculated with diphtheria toxoid. The following procedure is recommended: At 2 to 6 months of age, 2 adequate doses of diphtheria alum toxoid alone or combined with tetanus toxoid, 4 to 8 weeks apart; or 3 doses of combined pertussis vaccine and diphtheria toxoid (with or without added tetanus toxoid) administered at 3- to 5-week intervals; or 3 doses of fluid toxoid (fluid toxoid preferred in Great Britain and Canada). Whether with toxoid alone or combined with other antigens the basic course of inoculations must be reinforced by at least 1 recall or "booster" dose within 3 to 12 months. Reinforcing doses are essential in preschool life, desirable on entrance to school, and elective through school life and early adulthood. Where protection has been neglected in infancy, the program should be carried through as soon as the opportunity arises.
2. Adults subject to unusual risk, such as physicians, teachers, nurses, nursemaids, orderlies, and other hospital personnel, should receive diphtheria toxoid. It is desirable to identify susceptibles by a Schick test (0.1 ml intradermally containing 0.001 Lf of stabilized non-phenolized toxin). In order to serve as an adequate control of the Schick test and to reduce the hazard of severe local and constitutional reactions, an intradermal toxoid reaction test (0.1 ml. of a 1:100 dilution fluid toxoid in saline solution) is recommended. Schick-positive nonreactors to the control may be given toxoid in the usual dosage; persons who are Schick-positive and also reactors should be given small doses of suitably diluted purified toxoid containing a minimal amount of alum.
3. A majority of young adults of military age will become Schick-negative in the course of protection against tetanus, when inoculated with a special preparation containing diphtheria toxoid (about 2 Lf) in each 1 ml. of tetanus toxoid.

4. Pasteurization of milk supply.

5. Educational measures to inform the public and particularly the parents of little children of the hazards of diphtheria and the necessity and advantages of active immunization.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report obligatory in most states and countries, Class 2A (p. 5).
2. Isolation: Until 2 cultures from throat and 2 from nose taken not less than 24 hours apart fail to show diphtheria bacilli. Local or general application of antibiotic or chemotherapeutic agents invalidates the usefulness of bacteriological examination. Isolation may be terminated if the microorganism reported present is proved avirulent. Where termination of case by culture is impracticable, isolation may end with fair safety 14 days after onset. Where practicable, a virulence test should be made if throat cultures are reported positive 3 weeks or more after onset.
3. Concurrent disinfection: Of all articles in contact with patient, and all articles soiled by discharges of patient.
4. Terminal disinfection: Thorough airing and sunning of the sick room, with cleaning.
5. Quarantine: All intimate contacts, especially young children, should be kept under surveillance if found to be carriers or suffering from nasal discharge or sore throat. Adult contacts whose occupation involves handling of food or close association with children should be excluded from these occupations until shown not to be carriers by bacteriological examination.
6. Immunization of contacts: Child contacts less than 10 years of age, intimate exposed and not previously immunized with toxoid, may be given a prophylactic dose of antitoxin, 10,000 units, and at the same time a first dose of toxoid. Daily examination by a physician is advised for older children and adults, with such further active immunization as may be indicated; persons previously immunized should have a reinforcing dose of toxoid. Groups of persons, as in institutions, barracks, or other congested quarters, should have immediate Schick test, with toxoid reaction test, followed by active immunization of Schick-positives.
7. Investigation of contacts and source of infection: Search for unreported and atypical cases, carriers, and contaminated milk. Discovered carriers may be restricted and treated with various antibiotics. However, these control activities are of little importance in well immunized communities.
8. Specific treatment: If diphtheria is suspected, antitoxin should be given without awaiting bacteriological confirmation. The earlier antitoxin is given the more effective it is: 20,000 to 80,000 units depending upon duration of symptoms, area of involvement, and severity of the disease; and in a single dose after completion of sensitivity tests. Intramuscular administration usually suffices; in severe infections antitoxin both intravenously and intramuscularly is indicated. Bed rest is essential to minimize the hazard of cardiac and other complications. Sulfonamides are of no value. Penicillin should be used in conjunction with antitoxin but is not a substitute for antitoxin. Neither penicillin nor diphtheria antitoxin can be relied upon to shorten materially the period of communicability. Penicillin soaked compresses (500 units per ml) are of value in cutaneous diphtheria; also erythromycin and bacitracin.

C. Epidemic measures:

1. Immediate intensification of efforts to provide artificial immunization by diphtheria toxoid to the largest possible number of the population affected, with first and greatest emphasis upon protection of infants and preschool children.

D. International measures:

1. Active immunization of susceptible infants and young children traveling to or through countries where diphtheria is a common disease; a reinforcing dose of toxoid for those previously inoculated.
 2. Exchange of information between countries on current prevalence of diphtheria.
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DIPHYLLOBOTHRIASIS

1. **Identification**—An intestinal infection of man by an adult tapeworm. Symptoms are commonly trivial or absent. A few patients develop severe anemia; massive infections may be associated with toxic symptoms. A nonfatal infection of long duration. Synonym: Broad or Fish Tapeworm Infection.
Diagnosis is confirmed by identification in feces of eggs or proglottides (segments) of the worm.
2. **Occurrence**—Endemic in Finland, Baltic States, Russia, Siberia, Switzerland, Israel, Japan, Uganda, North America (Great Lakes region, eastern Canada, Alaska, and Florida), Argentina and Chile in South America, and in Australia. In some areas 10 to 30% of the population are infected, prevalence increasing with age. Persons in the United States become infected from eating or sampling uncooked infected fish from Middle West or Canadian lakes.
3. **Infectious agent**—*Diphyllobothrium latum*, also several other species.
4. **Reservoir and source of infection**—The main reservoir is an infected person discharging eggs in feces; other reservoir hosts include the dog, bear and other fish-eating mammals. The immediate source of infection is the flesh of infected fresh-water fish.
5. **Mode of transmission**—Man acquires the infection by eating parasitized fish, raw or inadequately cooked. Larvae developing in flesh of fresh-water fish are infective for definitive hosts. Eggs discharged from segments of worm must reach bodies of fresh water in which they mature, hatch, and produce infection in first intermediate host (copepods). Susceptible fish in turn ingest infected copepods and become second intermediate hosts.
6. **Incubation period**—From 3 to 6 weeks.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Man and other definitive hosts continue to disseminate eggs in the environment as long as worms remain in the intestine, possibly for several years.
8. **Susceptibility and resistance**—Man is universally susceptible. No apparent resistance follows infection.
9. **Methods of control**—

A. Preventive measures:

1. Prevention of stream and lake pollution by installation of disposal systems for human discharges in cities and villages, by chlorination

of sewage effluents, by sanitary privies in rural areas, and by education of the public.

2. Thorough cooking of fish or freezing for 24 hours at -10°C insures protection. Inspection of fish is not practical.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
2. Isolation: None.
3. Concurrent disinfection: None; sanitary disposal of feces.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Not usually a profitable procedure.
8. Specific treatment: Quinacrine or oleoresin of aspidium.

C. Epidemic measures: None.

D. International measures: None.

DRACONTIASIS

1. Identification—Infection with the nematode *Dracunculus medinensis*.

The female worm is about 1 meter long; when gravid it migrates from deep to subcutaneous tissue, usually of the leg. First manifestation of infection is when gravid female prepares to discharge larvae from uterus located at anterior end; burning and itching sensation at the point of exit, and frequently fever, nausea, vomiting, diarrhea, dyspnea, and generalized urticaria. Eosinophilia is usual. A vesicle forms at the point of exit, ruptures; uterus of worm protrudes and discharges milky fluid containing larvae. Careless attempts to extract the worm before uterus is empty may result in secondary bacterial infection. A nonfatal disease other than for septic complications. Synonym: Guinea Worm Disease.

Diagnosis is by microscopic identification of larvae or recognition of the adult worm after removal.

2. Occurrence—In India, Pakistan, Afghanistan, Russian Turkestan, Saudi Arabia, Iran, North and Central Africa, West Indies, and the Guianas. Local prevalence varies greatly; in some localities nearly all inhabitants are infected, in others very few. In North America, worms morphologically identical to *D. medinensis* occur in dogs, foxes, mink, and raccoons; no authentic indigenous case reported in man.

3. Infectious agent—*Dracunculus medinensis*, a nematode worm.

4. Reservoir and source of infection—Reservoir is an infected person, through a worm discharging larvae from the skin. The immediate source of infection is water from step-wells and ponds contaminated with infected *Cyclops*.

5. Mode of transmission—Larvae discharged into fresh water are swallowed by crustacea of the genus *Cyclops*; they penetrate into the body cavity and develop to the infective stage. Man swallows the *Cyclops* in drinking water, the larvae are liberated in the stomach or duodenum, migrate through the viscera, become adult and reach the subcutaneous tissues.

6. **Incubation period**—From swallowing of infected *Cyclops* to onset of symptoms is about 8 to 14 months.
7. **Period of communicability**—Until larvae have been completely evacuated from the uterus of the gravid worm, usually several days. Larvae may survive in water up to 6 weeks. Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—Susceptibility is universal. No acquired immunity; multiple and repeated infections occur in the same person.

9. **Methods of control**—

A. Preventive measures:

1. Provision of potable water. Abolition of step-wells and other measures to prevent contamination of drinking water by infected persons through immersion of affected parts.
2. Boiling of drinking water, or filtration through muslin cloth to remove *Cyclops*.
3. Control of *Cyclops* is by treatment of water at 6-week intervals with DDT wettable powder to give a concentration of 5 parts per million.
4. Education of the public to drink only boiled or filtered water. Instruction of infected persons in the mode of spread of the infection and the danger that exists in contaminating wells or other water supplies.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3C (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Information should be obtained as to source of drinking water at probable time of infection. Locality should be searched for other cases, and sources of drinking water should be examined microscopically for infected *Cyclops*.
8. Specific treatment: None.

C. Epidemic measures: In hyperendemic situations field survey to determine prevalence, to discover sources of infection, and to guide control measures as described in 9A.

D. International measures: None.

DYSENTERY, BACILLARY

1. **Identification**—An acute bacterial infection of the intestine characterized in severe cases by frequent stools containing blood, mucus and pus, and accompanied by malaise, toxemia, fever, cramps, and tenesmus. Many patients have mild infections with little or no diarrheal symptoms. Deaths are most frequent among infants, elderly debilitated persons, and those infected with Shiga bacillus; under other circumstances, the disease is rarely fatal. Synonym: Shigellosis.

Bacteriological diagnosis is by isolation of dysentery bacilli from feces or rectal swabs.

2. **Occurrence**—A significant portion of unreported diarrhea is bacillary dysentery. In England, Sonne infection is responsible for most recognized cases; in the United States, distribution of Flexner and Sonne strains is nearly equal, and Shiga infection is rare. Institutional outbreaks are frequent, with spread apparently by direct contact with unrecognized cases, or indirectly through nursing procedures and sometimes by food. Occurs in all parts of the world, arctic, temperate, and tropical. In the tropical and subtropical Orient, dysentery is a common and serious infection, occurring at all ages and causing many deaths, particularly of small children.
3. **Infectious agents**—Various species of genus *Shigella* (dysentery bacillus). *Sh. dysenteriae* (Shiga), *Sh. sonnei*, *Sh. flexneri*, *Sh. boydii*, and others.
4. **Reservoir and source of infection**—Reservoir is man; the source of infection is feces from an infected person.
5. **Mode of transmission**—By eating contaminated foods, or drinking contaminated water or milk, and by hand-to-mouth transfer of contaminated material; by flies; by objects soiled with feces of a patient or carrier.
6. **Incubation period**—One to 7 days, usually less than 4.
7. **Period of communicability**—During acute infection and until the infectious agent is absent from feces, usually within a few weeks even without specific therapy; with some strains of *Shigella* a few individuals become carriers for a year or two, rarely longer.
8. **Susceptibility and resistance**—Susceptibility is general but the disease is more common and more severe in children than in adults. Many inapparent, mild and unrecognized infections.

9. Methods of control—

A. Preventive measures:

1. Sanitary disposal of human feces.
2. Sanitary supervision of processing, preparation, and serving of all foods, particularly those moist and eaten raw; special attention to provision and use of hand-washing facilities. Protection of foods against contamination by flies.
3. Boiling of milk or pasteurization of milk and dairy products, see Typhoid Fever, 9A3 (p. 209).
4. Persons suffering from diarrhea should be excluded from handling food for public consumption, and from handling family food supply if possible.
5. Fly control and control of fly breeding, see Typhoid Fever, 9A6 (p. 210).
6. Protection and purification of water supplies, see Typhoid Fever, 9A1 (p. 209).
7. Reduction of general infant mortality in areas with high rates usually depends upon prevention of intestinal infection. Attention to the hygiene of breast feeding, scrupulous cleanliness in preparation, handling and refrigeration of food for children, boiling of milk and water for infant feeding, and continuous supervision of diet will contribute much to this aim. The suspicion of bacillary dysentery should enter into consideration of all acute diarrheal disease until proved otherwise by bacteriological examination of feces.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report obligatory in most states and countries, Class 2B (p. 5). Recognition and report of epidemics have more than usual importance in schools and institutions.
2. Isolation: During acute illness. Rigid personal precautions by attendants. Surveillance during carrier state, which is usually temporary, and prohibition from food handling.
3. Concurrent disinfection: Of feces and of articles soiled therewith. In communities with a modern and adequate sewage disposal system, feces can be disposed of directly into the sewer without preliminary disinfection.
4. Terminal disinfection: Cleaning.
5. Quarantine: Contacts should not be employed as food handlers during period of contact nor before repeated negative feces cultures are obtained.
6. Immunization of contacts: No known satisfactory method.
7. Investigation of contacts and source of infection: Search for unrecognized mild cases and convalescent carriers among contacts. For sporadic cases, such investigation is time consuming and gives meager results.
8. Specific treatment: The tetracyclines or chloramphenicol, by mouth or parenterally. Treatment should be continued for a minimum of 5 to 7 days. Sulfonamides with proper hydration have been recommended but the existence of drug-fast strains limits effectiveness.

C. Epidemic measures:

1. Groups of cases of acute diarrheal disorder should always be reported to local health authority at once, even in absence of exact identification of the disease or its cause.
2. Investigation of food, water, and milk supplies, general sanitation, and search for unrecognized mild cases and carriers.
3. An expected reduction in incidence after prophylactic administration of chemotherapeutic and antibiotic agents has not resulted when controlled experiments were undertaken.

D. International measures: None.

ECHINOCOCCOSIS

1. Identification—A disease caused by cysts (hydatids) formed in various tissues by the developing larvae of the tapeworm, *Echinococcus granulosus*. Liver and lung are most frequently involved, also kidney, pelvis, heart, bone, and central nervous system. Symptoms are variable. Fatality depends on site of cysts; they may give no symptoms through life, or in vital areas cause death. Synonyms: Hydatidosis, Echinococcosis.

Microscopic examination for hooklets, scolices, and cyst membranes in sputum, vomitus, urine, or feces after rupture of cysts, or in discharges from a sinus, aids in diagnosis. Eosinophilia may be present. Precipitin, complement-fixation, hemagglutination, and intradermal tests are other diagnostic aids. Confirmation is by examination of tissues obtained surgically or at autopsy.

2. **Occurrence**—The disease is relatively common in southern South America, eastern Australia, New Zealand, and Middle East countries. Hydatid infection is rare in England, Central Europe, and the United States, but relatively frequent in the Indian population of Canada and Eskimos in Alaska.
3. **Infectious agents**—*Echinococcus granulosus*, dog tapeworm; somatic infection with larvae only (hydatid disease) in man and numerous animal hosts, including sheep, cattle, pigs, camel, moose, caribou, and reindeer. A related species, *E. multilocularis* occurs in Central Europe, Siberia, North Pacific Islands, and Alaska.
4. **Reservoir and source of infection**—Reservoirs are carnivores infected with adult worms, especially the domestic dog, wolf, dingo, and to a lesser extent, other *Canidae*; these are the primary hosts. The source of infection is feces containing eggs of the parasite. The dog-sheep-dog cycle is important in endemic areas. The disease can be maintained in wild life.
5. **Mode of transmission**—By ingesting contaminated foods and water, and by hand-to-mouth transfer of eggs through contact with objects soiled with dog feces containing eggs. Eggs may survive for long periods in pastures, gardens, and around households. Ingested eggs hatch in intestine and larvae migrate to various organs to produce cysts.
6. **Incubation period**—Variable, from months to several years.
7. **Period of communicability**—Under natural conditions not transmissible from man to man nor from one intermediate host to another. Dogs begin to pass eggs of the parasite about 6 weeks after infection; in the absence of reinfection this ends in about 8 months.
8. **Susceptibility and resistance**—Man is universally susceptible. No apparent resistance follows infection.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Rigid control of slaughtering of herbivorous animals so that dogs do not have access to scraps of uncooked meat. Dogs are infected only by larval forms, hydatid cysts, present in food mammals.
 2. Licensing of dogs, with reduction in numbers in areas of endemic prevalence so far as may be compatible with occupational requirements. Mass anthelmintic treatment of dogs has varied effectiveness and must be repeated periodically.
 3. Education of school children and of the general public in endemic areas on the dangers of close association with dogs, and of the need for controlled slaughtering of animals.
 4. Incineration or deep burial of dead animals.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: In selected endemic areas; not a reportable disease in most states and countries. Class IB (pp. 5, 6).
 2. Isolation: None.
 3. Concurrent disinfection: None.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Examination of familial associates for suspicious tumors. Search for source of infection in dogs kept in and about the house.
 8. Specific treatment: None.

C. Epidemic measures:

1. In areas of high endemic prevalence, destruction of wild, vagrant, and nonessential dogs. Thorough treatment with anthelmintics of all essential dogs of the community twice yearly.
2. Systematic clinical, roentgenographic, and serologic examination of school children, farmers, hunters, and of others having close association with dogs.

D. International measures: Coordinated programs entered into by neighboring countries where the disease is endemic, designed to control the infection in animals and the movement of dogs from known enzootic areas.

ENTEROBIASIS

1. Identification—An exceedingly common nonfatal intestinal infection usually without symptoms. If severe, may cause pruritus ani with disturbed sleep and irritability, and local irritation from scratching. A variety of severe manifestations including appendicitis and salpingitis have been described, but their relationship is indefinite. Synonyms: Pinworm or Seatworm Disease, Oxyuriasis.

Diagnosis is by applying a scotch-tape applicator to the perianal region and examining microscopically for eggs; the material is best obtained in the morning before bathing or defecation.

2. Occurrence—Distribution is world-wide. An estimated 20% of the general population of the United States is infected. Prevalence is highest in children of school age, next highest in those of preschool age, and lowest in adults except for mothers of infected children who commonly have a high rate. Infection is characteristically familial. Crowding is an important factor; incidence is often high in institutions.

3. Infectious agent—*Enterobius vermicularis*, an intestinal round worm infecting only man. Eggs are infective within a few hours after leaving the gastrointestinal tract. After ingestion, eggs hatch in the stomach and small intestine; young worms mature in lower small intestine, cecum, and upper portions of colon. Gravid worms migrate to the rectum to discharge eggs on perianal skin; may migrate up genital tract of females and enter peritoneal cavity.

4. Reservoir and source of infection—Reservoir is infected persons, particularly children. Immediate sources of infection are clothing, bedding, food, or other articles contaminated with eggs of the parasite. Pinworms of animal hosts are not transmissible to man.

5. Mode of transmission—Direct transfer of infective eggs by hand from anal region to mouth of the same host, or indirectly to same host or new hosts through contaminated food and other objects. Dust-borne infection by inhalation is possible in heavily contaminated households.

6. Incubation period—The life cycle requires 3 to 6 weeks. Infections ordinarily build up from successive reinfections and are not recognized until several months.

7. Period of communicability—All infected persons are potential spreaders; in the absence of treatment for 2 to 8 weeks unless reinfected.

8. Susceptibility and resistance—Susceptibility is universal. Differences in frequency and intensity of infection are due to differences in exposure. No apparent resistance to repeated infections.

9. Methods of control—

A. Preventive measures:

1. Maintenance of clean facilities for defecation.
2. Insistence upon practice of personal hygiene of the toilet, particularly the washing of hands after defecation, and always before eating or preparing food.
3. Reduction of overcrowding in living accommodations; adequate provision of toilets and privies.
4. Bathing with sufficient frequency to keep the body clean; use of clean underclothing, night clothes, and bed sheets at frequent intervals.
5. Habits of nail biting and scratching bare anal area should be discouraged.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6). School authorities should be advised of school outbreaks.
 2. Isolation: None.
 3. Concurrent disinfection: Sanitary disposal of feces and washing of hands in soap and water after defecating and before eating. Change bed linen and underwear of infected person daily. Eggs on discarded linen are killed by exposure to temperatures of 55° C (132° F) for a few seconds, either by boiling or by the usual household washing machine.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Each infected individual is a source of infection. All members of an affected family or institution should be examined.
 8. Specific treatment: Piperazine salts, cyanine compounds (dithiazanine and vinquin), gentian violet medicinal. All infected members of a household or institution should be treated simultaneously.
- C. Epidemic measures: Outbreaks in schools and institutions require strict hygienic measures and cleanliness. Toilet seats should be washed daily with disinfectant. Control measures should include facilities for treatment.

D. International measures: None.

FASCIOLOPSIASIS

1. Identification—An infection of the small intestine with the trematode or fluke, *Fasciolopsis buski*. Symptoms are from absorption of toxin rather than direct blood destruction. Severity depends on number of worms and the general state of health; death is rare. The large flattened red worms attach to mucous membranes of the small intestine, particularly duodenum. Diarrhea of a nontyphoid type usually alternates with constipation; vomiting and anorexia are frequent. Acute intestinal obstruction may occur with large numbers of worms. Patients may show edema of face within 20 days after infection, also of abdominal

wall and legs; ascites is common. Eosinophilia is usual, and secondary anemia occasional.

Diagnosis depends on finding worms or characteristic eggs in feces. Eggs are easily detected because of their large size; occasionally worms are vomited.

Differentiation is required from eggs of *Fasciola hepatica*, the fluke commonly infecting the bile ducts of sheep, cattle, and other ruminants throughout the world. The disease, fascioliasis, is frequently reported in man in Cuba.

2. **Occurrence**—Widely distributed in the Orient, including Central and South China, Formosa, Bengal, Assam, Thailand, Tonkin, Annam, Sumatra, and Borneo. Prevalence is often extremely high.
3. **Infectious agent**—*Fasciolopsis buski*.
4. **Reservoir and source of infection**—Man, pig, and dog are definitive reservoir hosts of adult flukes. Aquatic plants eaten raw are the source of infection to man; in China, particularly, the nuts of the red water-caltrop grown in enclosed ponds, and tubers of the so-called "water chestnut," as the hull or skin is peeled off with the teeth and lips.
5. **Mode of transmission**—Eggs passed in feces develop within 3 to 7 weeks under favorable conditions; miracidia hatch, penetrate snails (*Segmentininae* of the *Planorbidae*) as intermediate host; cercariae develop, are liberated and encyst in aquatic plants which transmit the infection to man. Period of development from infection of snail to development of encysted infective metacercariae is 7 to 8 weeks.
6. **Incubation period**—About 3 months, from ingestion of infective larvae until passage of eggs.
7. **Period of communicability**—As long as viable eggs are discharged by patient; without treatment, probably for many years. Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—Susceptibility is universal; in undernourished individuals the ill effects are pronounced.

9. Methods of control—

A. Preventive measures:

1. Education of people in endemic areas regarding life cycle of the parasite.
2. Treatment of nightsoil before use as fertilizer; chemical treatment (unslaked lime, 1 part to 1,000 parts of water) or long storage destroys eggs, as do drying, freezing, or heating.
3. Drying the particular plants involved, or if eaten fresh, dipping them into boiling water for a few seconds; both methods kill metacercariae.
4. Destruction of snail intermediate hosts.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3C (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: Sanitary disposal of feces.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.

7. Investigation of contacts and source of infection: In the individual case, of little value. A community problem (see 9C below).
 8. Specific treatment: Tetrachlorethylene and hexylresorcinol crystalloids have been used with considerable success.
- C. Epidemic measures:** Locate those aquatic plants eaten fresh, which carry encysted metacercariae; identify infected snails in water with plants and prevent access of human feces to the water.
- D. International measures:** None.

FILARIASIS

1. **Identification**—A nematode infection with early acute manifestations of fever, lymphadenitis, retrograde lymphangitis of extremities, orchitis, epididymitis, funiculitis, and abscess. These are primarily allergic reactions, but secondary bacterial infection may occur and lead to the occasionally recorded death. After prolonged or repeated infection, obstruction to lymph flow often leads to hydrocele and elephantiasis of limbs, genitalia or breasts, or to chyluria. Female worms give rise to larvae, microfilariae, which in the absence of lymphatic obstruction, reach the blood stream. Many infected persons have no clinical manifestations but do have circulating microfilariae; many persons with clinical manifestations do not have circulating microfilariae. A nocturnal periodicity of microfilariae in the peripheral blood (10 p.m.–2 a.m.) occurs in all endemic areas except in those Pacific Islands where *Aedes* mosquitoes are the vectors.
Microfilariae are best detected in blood specimens taken at optimum daily periodicity, and examined in thick-film preparation or in stained sediment of laked blood. Skin test is nonspecific, of value in absence of microfilaremia.
2. **Occurrence**—Filariasis due to *W. bancrofti* is endemic in the West Indies, coastal Central America, northern and eastern South America, Saudi Arabia, Madagascar, India, Southeast Asia, China, Korea, Japan, northern Australia, and most of the Pacific Islands. *W. malayi* is endemic only in Southeast Asia, India, Central China, and a few islands of Indonesia. Local foci of high prevalence are often surrounded by nonendemic areas. High prevalence depends upon a large reservoir of infection and abundant vector breeding.
3. **Infectious agents**—Nematode worms, *Wuchereria bancrofti* and *W. malayi*.
4. **Reservoir and source of infection**—Reservoir is man, with microfilariae in the blood. Immediate source of infection is a mosquito.
5. **Mode of transmission**—By bite of mosquitoes harboring infective larvae. *W. bancrofti* is transmitted in nature by many species, the most important *Culex fatigans*, *C. pipiens*, *Aedes polynesiensis* (pseudoscutellaris), and several species of *Anopheles*. *W. malayi* is transmitted by various species of *Mansonia* and *Anopheles*. The microfilariae penetrate the stomach wall of the mosquito, lodge in thoracic muscles, develop into infective larvae, migrate to the proboscis, and are transmitted to the new host as the infected mosquito bites.
6. **Incubation period**—Allergic manifestations may appear as early as 3 months after infection. Microfilariae do not appear in the blood until at least 9 months.

7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Man remains infective for mosquitoes as long as microfilariae are present in the blood, which may be years. Communicability in the mosquito is from at least 10 days after infective feed until all infective larvae are discharged.
8. **Susceptibility and resistance**—All persons are probably susceptible. Repeated infection apparently occurs in endemic regions. Acquired immunity is unknown.
9. **Methods of control**—

A. Preventive measures:

1. Determine by dissection the vector or vectors in each locality; study times and places of feeding and locate breeding places. In most areas other than the South Pacific, attack adult mosquitoes by spraying buildings with an acceptable residual insecticide (DDT or other), screening of houses, use of bed nets, and insect repellents. Attack larvae by eliminating small breeding places and treating others with larvicides. Each local situation requires individual study.
2. Education of the public concerning mode of transmission, and methods of mosquito control.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic regions; in most countries not a reportable disease, Class 3C (pp. 5, 6). Reporting of cases with demonstrated microfilariae provides data on potential transmission. Cases of elephantiasis without microfilariae in the blood should not be reported as filariasis, but are usefully recorded in estimating prevalence or in planning control programs.
2. Isolation: Not practicable. So far as possible patients with microfilariae in blood should be protected from mosquito bites as a means of reducing transmission.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Only as a part of a general community effort. (See 9A and 9C.)
8. Specific treatment: Diethylcarbamazine (hetrazan®) results in rapid disappearance of most or all microfilariae from the blood, but may not kill or even sterilize the adult female worm; microfilariae usually reappear after several months. Sodium thiacetarsamide (Caparsolate sodium) causes slow disappearance of microfilariae during treatment without subsequent increase over a 2-year period. Action of this compound apparently is against adult worms rather than microfilariae.

C. Epidemic measures: The first essential in a program of control in areas of high endemicity is an appraisal of the local situation, particularly the bionomics of mosquito vectors, prevalence and incidence of disease, and environmental factors responsible for transmission. Vector control is the fundamental approach. Even partial control by anti-mosquito measures may reduce incidence and restrict the endemic focus. Measurable results are slow because of the long incubation period. Mass treatment of known infected persons with hetrazan contributes materially.

- D. International measures:** Coordinated programs entered into by neighboring countries where the disease is endemic with the purpose of limiting migration of infected persons across international boundaries, and instituting treatment and other control measures near such boundaries.

FOOD POISONING, STAPHYLOCOCCAL

Food poisoning is a generic term for disease of man characterized by gastroenteritis of abrupt evolution, acquired through food, having a characteristic grouping of cases, and arising either from intoxication or infection. Numerous organic and inorganic substances act as agents of intoxication. Microorganisms produce bacterial toxins by growth in food to give subsequent intoxication and others act through infection. Certain foods are of themselves poisonous; for example, poisonous fishes such as the Moray eel, puffer and others.

Food poisoning is distinguished from food-borne infection. The effects of food poisoning are promptly evident and the amount of the particular food ingested has a relation to severity, suggesting the importance of preformed elements. Food-borne infection with a number of intestinal pathogens, with streptococci and with the agents of diphtheria, tuberculosis, and undulant fever, follows a usual incubation for the particular disease, and clinical course and manifestations are not as a rule materially altered by the circumstance of food serving as the vehicle of infection.

The interest here is in intoxications of bacterial origin. In addition to a wide range of activities in general and local infections (see p. 173), staphylococci grow in food, producing a toxin that gives rise to food poisoning. *Clostridium perfringens*, the Welch bacillus, has a similar capacity less frequently evidenced. Botulism (p. 44) is likewise a food poisoning but the clinical disease is of the central nervous system rather than of the gastrointestinal tract. *Salmonellae* (p. 162) produce a food poisoning, more an infection than an intoxication, among a variety of other pathogenic activities. The description of staphylococcal food poisoning follows:

- 1. Identification**—A poisoning (not an infection) of abrupt and sometimes violent onset with severe nausea, vomiting, and prostration; often severe diarrhea. Deaths are exceedingly rare. Diagnosis is usually on the basis of grouping of cases and short interval between eating food and onset of symptoms.
Isolation of large numbers of staphylococci from suspected food permits presumptive diagnosis. Demonstration of ability of the organism to produce enterotoxin is essential to confirmation. Phage typing may aid in epidemiologic investigations.
- 2. Occurrence**—Widespread and a relatively frequent disease; the principal acute food poisoning in the United States.
- 3. Toxic agent**—Enterotoxin of certain strains of staphylococci; the toxin is stable at boiling temperature. Staphylococci multiply in food, producing toxin which causes poisoning.
- 4. Reservoir and source of poisoning**—Source of contamination of the food in most instances not determinable; believed to be ordinarily of human origin.
- 5. Mode of transmission**—Custard filled pastry is the most common vehicle; processed meats, especially ham, are responsible for some outbreaks; also milk from cows with specifically infected udders. Dried milk powder has been involved with increasing frequency in recent years.

6. **Incubation period**—Interval between taking food and onset of symptoms is one-half hour to 4 hours, usually 2 to 4 hours.
7. **Period of communicability**—Not applicable.
8. **Susceptibility and resistance**—Most persons are susceptible, although individual reactions are variable.
9. **Methods of control**—

A. Preventive measures:

1. Prompt refrigeration of sliced and chopped meats and of custards and cream fillings to avoid multiplication of staphylococci accidentally introduced; fill pastries with custard just prior to sale, or subject finished product to adequate heat treatment. Immediate disposal or proper refrigeration of leftover foods.
2. Some health departments have forbidden sale of custard-filled products during summer months.
3. Temporary exclusion from food handling of persons suffering from pyogenic skin infections, especially of the hands.
4. Education of food handlers in strict attention to sanitation and cleanliness of kitchens, including refrigeration, handwashing, attention to the fingernails, and the danger of working while having skin infections.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory report of epidemics; also suspected or confirmed cases with evidence of grouping or association with others in time and place, Class 4 (p. 6).
2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Not applicable.
7. Investigation of contacts and source of infection: See 9C.
8. Specific treatment: None.

C. Epidemic measures:

1. Search for food contaminated with staphylococci and for food handlers with skin infections or the nasal carrier state.
2. Destruction of any remainder of contaminated food after samples have been taken for laboratory examination.

D. International measures: None.

GLANDERS

1. **Identification**—Glanders is a highly communicable disease of solipeds (horses, mules, donkeys), but rare in man. Two clinical forms occur, an infection of the skin known as farcy and a generalized infection known as glanders. Both may be acute or chronic, chronic infection being more common in animals and acute infection in man. Infection of man may be clinical or inapparent; symptoms vary widely. Infections originating in the skin are characterized by a nodule at the site of inoculation, a surrounding area of lymphangitis and swelling, a generalized papular and sometimes pustular eruption, and broncho-

pneumonia; highly fatal, often within 8 to 10 days. Primary infections of nasal mucosa tend to resemble chronic glanders of the horse but are more often fatal, with ulcers of mucosa, regional lymphangitis and adenitis, and general dissemination of infection including skin lesions and joint manifestations. Laboratory infection through inhalation of infectious material produces pneumonitis resembling viral pneumonia.

Diagnosis is by complement-fixation test, positive in the third week; by agglutination test with rise in antibodies in the second week; by late appearance of mallein reaction; and by bacteriological culture and isolation of the organism from lesions of experimentally infected hamsters and guinea pigs.

Melioidosis is another rare disease, to be differentiated from glanders. The acute form is characterized by generalized caseous or purulent abscesses giving rise to septicemia and death in 1 to 4 weeks. The chronic form is characterized by discharging sinuses from bone or deep skin lesions, and may last for years; inapparent forms likely exist. The infectious agent is *Pseudomonas pseudomallei*. Sporadic cases have been reported in many parts of the world, including continental United States; exists mainly in Southeast Asia, also Central and South America, where local climatic conditions favor survival of the infectious agent. Transmission presumably occurs through contact of overt or inapparent skin wounds with contaminated water. Identified in rats and in sheep and other domestic animals but their role in the epidemiology of the disease has not been determined. Under natural conditions not directly transmissible from man to man.

2. **Occurrence**—Rare and sporadic; almost exclusively in occupations bringing contact with animals, such as veterinarians or others treating glandered animals or conducting autopsies, laboratory workers and animal attendants. Of decreasing world frequency as mechanized transport replaces horse-drawn vehicles; still observed in Asia, Africa, eastern Europe, and South America. At long intervals cases are seen in North America and western Europe.
3. **Infectious agent**—*Actinobacillus mallei*, glanders bacillus.
4. **Reservoir and source of infection**—Reservoirs are horses, mules, donkeys, and man. Sources of infection are discharges from open lesions of the respiratory tract or exudates of skin lesions.
5. **Mode of transmission**—Infection in man is through inoculation of skin by direct contact with diseased animals, their tissues, cultures of the organism, or in handling artificially infected laboratory animals; to a lesser extent by indirect contact with articles soiled with discharges. Hand-to-mouth transmission and infection by ingestion also occur. Rarely transmitted from man to man among attendants caring for patients. Laboratory infections are relatively frequent, by inoculation of skin or air-borne by inhalation of moist particles containing the infectious agent.
6. **Incubation period**—Variable; usually 1 to 5 days.
7. **Period of communicability**—Until organisms disappear from discharges or until lesions have healed.
8. **Susceptibility and resistance**—Man apparently has a high grade inherent insusceptibility. Despite close association with animals where half or more are infected, cases in man are few. In this respect glanders is like foot and mouth disease, where authenticated infection in man is highly uncommon and existing areas of frequency much overrated.

9. Methods of control—

A. *Preventive measures:*

1. Prevention of glanders in man depends primarily upon control of glanders in equine species. In areas of endemic prevalence, measures include abolition of common feeding and drinking troughs for horses, sanitary supervision of stables and other places where horses are kept, and annual mallein testing of all horses and members of the horse family; also when offered for sale.
2. Extreme care should be practiced by all laboratory workers handling cultures of this organism or artificially infected laboratory animals, their cages, and materials with which they have come in contact.

B. *Control of patient, contacts, and the immediate environment:*

1. Report to local health authority: Case report obligatory in most states and countries, Class 2A (p. 5). Report also to local livestock sanitary or agriculture authority.
2. Isolation: At home or hospital; attendants should exercise scrupulous care to avoid contact with skin lesions or nasal secretions.
3. Concurrent disinfection: Discharges from patients and articles soiled therewith.
4. Terminal disinfection: Thorough cleaning.
5. Quarantine: None.
6. Immunization of contacts: No effective procedure.
7. Investigation of contacts and source of infection: Human carriers are unknown. The problem is of animals (see 9C).
8. Specific treatment: Experience is limited, but effective results are reported with sulfadiazine; penicillin or erythromycin may be useful.

C. *Epidemic measures:*

1. Not applicable to man, a sporadic disease.
2. Principal concern is with outbreaks among horses and other solipeds, which may be widespread or local. Quarantine of all animals in a contaminated stable until all have been mallein tested and retested, infected animals removed, and stable, materials and surroundings disinfected. Destruction of infected animals is essential. Isolate newly arrived animals until freedom from glanders is assured by negative mallein test.

D. *International measures:* None.

GNOCOCCAL INFECTION

Urethritis and salpingitis of adults, vulvovaginitis of children, and ophthalmia of newborn and adults are inflammatory conditions caused in common by gonococci. All are maintained in a population by reason of the continued presence of gonorrhea; together they constitute an epidemiologic entity and similar principles of control apply to the group.

Clinically indistinguishable infections of the same anatomic structures are caused by a number of other infectious agents. This presentation relates specifically to gonococcal disease but the final paragraph under identification of each of the three gonococcal conditions gives the general characteristics of these other infections. They occur frequently and methods of control are often ill-defined.

A. GONOCOCCAL URETHRITIS (GONORRHEA)

1. Identification—A more or less self-limited infectious disease of venereal origin initiated by the gonococcus, an organism having affinity for columnar and transitional epithelium. The disease is thereby limited to structures where such tissues are found. Accordingly, gonorrhea in the male and female differ in course, in seriousness and in ease of identification. In males, a thick yellow purulent discharge of the anterior urethra usually appears 3 to 9 days after effective exposure; infection may extend to the posterior urethra and after varying intervals result in epididymitis, prostatitis, arthritis, and endocarditis. In females, the disorder is in three stages: (1) a few days after exposure, an initial urethritis or cervicitis, often so mild as to pass unnoticed; (2) a stage of pelvic invasion, usually accompanying the first, second, or later menstrual period after infection, with mild or severe symptoms of salpingitis or pelvic peritonitis; (3) a stage of residual and often chronic infection. Death from gonorrhea is rare, but early and late manifestations, especially complications, are common and seriously incapacitating.

Bacteriologic culture is requisite to diagnosis of gonorrhea in the female, and desirable in gonorrhea of the male.

Widespread and frequent nongonococcal urethritis, possibly also of sexual origin, seriously complicates the clinical diagnosis of gonorrhea in the male. In some countries the incidence exceeds that of gonorrhea and the condition is reportable. It is notoriously resistant to treatment, and much is unknown of etiology, communicability, clinical course, and control; it may be initiated by a number of infectious agents.

- 2. Occurrence**—World wide and a common disease, particularly among persons of lower economic status; affects both sexes and practically all ages, especially the age groups of greatest sexual activity. Lack of accurate diagnosis and incomplete reporting preclude reliable estimates of incidence.
- 3. Infectious agent**—*Neisseria gonorrhoeae*, the gonococcus.
- 4. Reservoir and source of infection**—Man is the only reservoir. Source of infection is exudates from mucous membranes of infected persons.
- 5. Mode of transmission**—Almost wholly by sexual intercourse, even in children; occasionally, in the newborn, by transfer from an ophthalmic infection acquired during birth; and in institutions for children, by careless and indiscriminate use of rectal thermometers.
- 6. Incubation period**—Usually 3 to 9 days, sometimes longer.
- 7. Period of communicability**—For months or years unless interrupted by specific therapy, which ends communicability within hours or days.
- 8. Susceptibility and resistance**—Susceptibility is general. Spontaneous recovery is usual in the absence of reinfection. Acquired immunity has not been demonstrated; one attack does not protect against subsequent infection.

9. Methods of control—**A. Preventive measures:**

1. Except for measures applying specifically to gonorrhea, primarily the use of chemoprophylactic agents in the eyes of the newborn (9A1, p. 87) and special attention (abortive treatment) to female contacts of male patients (9B7, infra), the preventive measures are those of Syphilis (see 9A, p. 187).

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report is required in many states and countries, Class 2B (p. 5).
2. Isolation: None; antibiotics promptly render discharges noninfectious. Refrain from sexual intercourse with untreated previous sexual partners to avoid reinfection.
3. Concurrent disinfection: Care in disposal of discharges from lesions and articles soiled therewith.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Not applicable (see 9B7).
7. Investigation of contacts and source of infection: Interview of patients and tracing of contacts are the fundamental features of a program for control. Trained interviewers obtain the best results. Female contacts of male patients with gonorrhea should be treated at once, on epidemiologic grounds only, because of delays and difficulties in diagnosis and the practical fact that sexual exposure continues despite advice to the contrary. Include all sexual contacts of 10 days prior to onset; also examine them serologically for syphilis over a period of several weeks.
8. Specific treatment: Procaine penicillin or other penicillin preparations (PAM or benzathine penicillin G) in 1 intramuscular injection on clinical, laboratory or epidemiological grounds. Streptomycin is used in case of sensitivity or resistance to penicillin.

C. Epidemic measures: Intensification of routine procedures, especially therapy on epidemiologic grounds.

D. International measures: See Syphilis 9D, p. 188.

B. GONOCOCCAL VULVOVAGINITIS OF CHILDREN

1. **Identification**—An inflammatory reaction of the urogenital tract of pre-pubescent females, characterized by redness and swelling of mucous membrane and mucopurulent discharge of varying degree; in severe infections, excoriation of labia and thighs and extension to urethra and bladder. A self-limited disease in that more than three-fourths of patients recover spontaneously within 3 to 6 months; a carrier state sometimes persists.

Diagnosis is established by bacteriologic culture of exudates; stained smears are unreliable.

Gonococcal vulvovaginitis is to be differentiated from acute vulvovaginitis due to a variety of other infectious agents. In the United States, only about 25% of cases of vulvovaginitis are of gonococcal origin. Clinical manifestations of the several diseases are usually indistinguishable and recognition is by bacteriological means. Outbreaks of nongonococcal proctitis and vulvovaginitis occur in institutions for children.

2. **Occurrence**—Extent not known but presumably widespread, particularly in families of lower social and economic levels where standards of personal, sexual, and general hygiene are low. Epidemics are most frequent in institutions for children.
3. **Infectious agent**—*Neisseria gonorrhoeae*, gonococcus.
4. **Reservoir and source of infection**—The reservoir is man. The source of infection is exudates from infected persons; infrequently, contaminated moist articles. The existence of transient inapparent gonococcal infection among children has been demonstrated.

5. **Mode of transmission**—Intimate direct contact with adult patients in the home, willing and unwilling sexual contact, and insertion of contaminated instruments and foreign bodies in vagina and rectum.
6. **Incubation period**—Usually 3 to 9 days.
7. **Period of communicability**—While discharges persist, usually 3 to 6 months; communicability may continue after clinical manifestations cease.
8. **Susceptibility and resistance**—Susceptibility is related to type of epithelium lining vagina; until puberty: columnar or transitional epithelium; after puberty: stratified squamous type, not attacked by gonococci. Susceptibility is general among female children; the frequency of spontaneous recovery is proof of a developing resistance. One attack does not protect against subsequent infection.

9. Methods of control—

A. Preventive measures:

1. Fundamentally dependent on control of gonorrhea (see p. 84); general measures are those of Syphilis (see 9A, p. 187).
2. Proper supervision of institutions for children, with rigid enforcement of hygienic principles and realization that sex education must begin earlier in life than commonly believed.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report is required in most states and countries, Class 2A (p. 5).
2. Isolation: Until 24 hours after administration of antibiotics.
3. Concurrent disinfection: Care in disposal of discharges from lesions and articles soiled therewith.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None; chemoprophylaxis on suspicion of infection.
7. Investigation of contacts and source of infection: The proved importance of sexual transmission among children calls for trained interviewers to elicit history of sexual contact among playmates, family members and older males within the family group and outside. Histories are unreliable; actual search for gonorrhea among persons in the environment of the child is necessary.
8. Specific treatment: Penicillin in a single repository dose.

C. Epidemic measures:

1. Prompt search for source of infection within the institution or group affected and introduction of measures to protect preadolescent girls.
2. Education of those in charge of children as to causes of outbreaks, and the sources and development of the disease, with special emphasis upon personal cleanliness of children. The importance of probable sexual transmission should be emphasized.

D. International measures: None.

C. GONOCOCCAL OPHTHALMIA NEONATORUM

1. **Identification**—Acute redness and swelling of the conjunctiva of one or both eyes, with mucopurulent or purulent discharge, in which gonococci are identifiable by microscopic and cultural methods.

Gonococcal ophthalmia neonatorum is only one of a number of acute inflammatory conditions of the eye or of the conjunctiva occurring

within the first 3 weeks of life, and collectively known as ophthalmia neonatorum. Differentiation is only by bacteriological means. The gonococcus is the most important but not necessarily the most frequent among infecting agents, which include meningococci, hemophilic bacilli, a virus (inclusion blennorrhoea), and others. All purulent inflammation of the conjunctiva of the newborn is to be regarded as gonococcal until proved otherwise. Synonyms: Gonorrheal Ophthalmia, Acute Conjunctivitis of the Newborn.

2. **Occurrence**—Varies widely according to measures for the prevention of infection of the eyes of the newborn by attendants at delivery. Infrequent where care of the newborn is adequate; globally, the disease is still an important cause of blindness.
3. **Infectious agent**—*Neisseria gonorrhoeae*, gonococcus.
4. **Reservoir and source of infection**—Man is the only reservoir. Source of infection is the maternal birth canal of an infected person.
5. **Mode of transmission**—Contact during childbirth.
6. **Incubation period**—Usually 36 to 48 hours.
7. **Period of communicability**—For 24 hours following specific treatment or lacking that until discharges from infected membranes have ceased.
8. **Susceptibility and resistance**—Susceptibility is general. Immunity to subsequent gonococcal infection does not follow attack.
9. **Methods of control**—

A. Preventive measures:

1. Use of an established effective preparation for protection of the eyes of babies at birth, preferably instillation of silver nitrate solution. It is recognized that a specific and stable antibiotic agent which remains *in situ* long enough to be effective (for example, penicillin ointment 100,000 units per gram) appears to be as effective as silver nitrate solution. However, such preparations have not been used in large-scale, long-term controlled studies in areas where the disease is prevalent. Hence 1% silver nitrate solution stored in individual wax capsules remains the preferred prophylactic agent for general use.
2. Depends fundamentally on control of gonorrhea (see p. 84), especially diagnosis and treatment of gonorrhea of the mother during prenatal period; general preventive measures are those of Syphilis (see 9A, p. 187).

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report is required in most states and countries, Class 2A (p. 5).
2. Isolation: For the first 24 hours after administration of antibiotic.
3. Concurrent disinfection: Care in disposal of conjunctival discharges and articles soiled therewith.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Not applicable; prompt treatment on recognition or clinical suspicion of infection.
7. Investigation of contacts and source of infection: Examination and treatment of mother and her consort.
8. Specific treatment: Parenteral penicillin.

C. Epidemic measures: None; a sporadic disease.

D. International measures: None.

GRANULOMA INGUINALE

1. **Identification**—A mildly communicable, nonfatal, chronic, and progressively autoinoculable infection of skin and mucous membrane of the external genitalia, also occurring extragenitally and occasionally involving regional lymphatics. A small nodule, vesicle or papule becomes a creeping, exuberant, ulcerative, or cicatricial process of the skin, frequently painless and extending peripherally with characteristic rolled edges and formation of fibrous tissue, often with active granulomatous tissue. New lesions may occur by autoinoculation and coalesce with older ones. Local swelling may precede lesions of the inguinal region. Extragenital lesions have a predilection for warm and moist surfaces such as folds between scrotum and thighs or labia and vagina. If neglected, the disease may result in serious destruction of genital organs and spread to other parts of the body. Synonyms: Donovaniasis, Ulcerating Granuloma, Granuloma Pudendi Tropicum, Granuloma Contagiosa, Chronic Venereal Sore, and Granuloma Venereum.

Laboratory confirmation is by demonstrating the causative organism and the pathognomonic cell in stained spreads of punch biopsy tissue taken from lesions, and by histologic examination of biopsy specimens.

2. **Occurrence**—An infrequent disease of tropical, subtropical, and temperate areas both north and south of the equator; apparently more frequent among males than females, and among groups of lower social status. Occurs predominantly at ages of from 20 to 40 years.
3. **Infectious agent**—*Donovania granulomatis*, the Donovan body.
4. **Reservoir and source of infection**—Reservoir is man. Source of infection is probably the active lesions of infected persons.
5. **Mode of transmission**—Unknown; venereal transmission is unproved but presumably by direct contact during sexual intercourse.
6. **Incubation period**—Unknown; presumably between 8 days and 12 weeks.
7. **Period of communicability**—Unknown; likely during open lesions of skin or mucous membrane.
8. **Susceptibility and resistance**—Susceptibility is variable. Immunity apparently does not follow attack.

9. Methods of control—

A. Preventive measures:

1. Preventive measures are currently those for venereal diseases in general. (See Syphilis, 9A, p. 187.)

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas (USA, some states); not a reportable disease in most states and countries. Class 3B (pp. 5, 6).
2. Isolation: None; avoid close personal contact with others until lesions are healed.
3. Concurrent disinfection: Care in disposal of discharges from lesions and articles soiled therewith.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Not applicable; prompt treatment on recognition or clinical suspicion of infection.
7. Investigation of contacts and source of infection: Examination of sexual contacts with special emphasis on marital partners.

8. **Specific treatment:** Streptomycin, tetracycline antibiotics, and chloramphenicol are effective; recurrence is to be expected in some instances, usually responding to a second course. Antimony compounds are effective but healing is slow and therapy must be continued for some months after apparent recovery.

C. Epidemic measures: Intensification of preventive and control activities.

D. International measures: See Syphilis 9D, p. 188.

HEPATITIS, INFECTIOUS

1. **Identification**—An acute infection characterized by fever, anorexia, nausea, malaise, and abdominal discomfort, followed by jaundice. Leucopenia is usual, and bile may be detected in urine. Convalescence may be prolonged. Many infections are mild, without jaundice, and recognizable only by liver function tests; fulminating and usually fatal cases of acute yellow atrophy of liver are rare. Usually a benign disease; in epidemics fatality rarely exceeds 5% and commonly is nearer 2%. Synonyms: Epidemic Hepatitis, Epidemic Jaundice, Catarrhal Jaundice. No specific laboratory tests available.
2. **Occurrence**—World-wide, sporadically, and in epidemics, with outbreaks most commonly in institutions, in rural areas, and in military forces during wars. Incidence in rural areas higher than in cities. Most common among children and young adults, less with advancing years. Incidence highest in autumn and early winter in temperate zones.
3. **Infectious agent**—Virus of infectious hepatitis.
4. **Reservoir and source of infection**—Man is the reservoir. Sources of infection are feces and blood from infected persons. Presence of virus in nose and throat discharges not proved but commonly assumed on epidemiologic grounds.
5. **Mode of transmission**—Probably through intimate person-to-person contact, with respiratory spread possible; also through transfusion of whole blood, injection of blood serum or plasma from infected persons, and by accidental contamination of syringes or needles with traces of blood from such persons; transmissible to human volunteers by ingestion or parenteral inoculation of blood or filtered fecal suspensions from patients in the acute phase. Epidemics have been related to contaminated water, food, and milk.
6. **Incubation period**—Long and variable, from 10 to 40 days, commonly 25 days.
7. **Period of communicability**—Unknown; virus demonstrated in blood before clinically recognized disease; evidence of fecal carrier state of at least 5 to 15 months. Clinical experience suggests greatest communicability from a few days before to a few days after onset, usually not exceeding 7 days.
8. **Susceptibility and resistance**—Susceptibility is general. Degree and duration of immunity after attack unknown but probably lifelong. No animal other than man is known to be susceptible.
9. **Methods of control**—
 - A. Preventive measures:**
 1. Good sanitation and personal hygiene, with particular emphasis on disposal of feces.

2. Proper technical procedures to prevent transmission by blood or blood products from an infected donor or through use of improperly sterilized syringes and needles. (See Serum Hepatitis, 9A, p. 91).

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Reporting advisable though not required in many states and countries, Class 3B (pp. 5, 6).
2. Isolation: During first week of illness.
3. Concurrent disinfection: Feces, and nose and throat secretions.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Immune serum globulin, 0.01 ml per pound of body weight intramuscularly and promptly after exposure, gives passive protection even as late as 6 days before onset of disease and lasting 6 to 8 weeks. Active immunity may result from infection rendered inapparent through protection by immune globulin.
7. Investigation of contacts and source of infection: Search for missed cases and surveillance of contacts.
8. Specific treatment: None.

C. Epidemic measures:

1. Epidemiologic investigation to determine possible transmission by water, blood or blood products.
2. Special efforts to improve sanitary and hygienic practices, to reduce fecal contamination of foods and water and careless disposal of nose and mouth discharges.
3. Focal concentration of disease in schools and institutions of limited populations may suggest mass prophylaxis with immune serum globulin.

D. International measures: None.

HEPATITIS, SERUM

1. **Identification**—Clinically indistinguishable from infectious hepatitis (p. 89). Chief differences are longer incubation period, under natural conditions a lack of direct transmission from man to man, and a higher fatality, varying from 6 to 12%. Synonym: Homologous Serum Jaundice.

Absence of naturally occurring infection among associates and history of injection of blood products 2 to 6 months previously are essential to differentiation from infectious hepatitis.

2. **Occurrence**—World-wide in distribution. Incidence among recipients of pooled blood products varies from 2 to 15%, and with known iatrogenic plasma as high as 60%.
3. **Infectious agent**—Virus of serum hepatitis.
4. **Reservoir and source of infection**—Man is the reservoir. Sources of infection are blood or blood products from an infected person.
5. **Mode of transmission**—By parenteral (intravenous, intramuscular, or subcutaneous) inoculation of infected human blood, plasma, serum, or thrombin; or by use of syringes or needles contaminated with traces of blood from an infected person. Immune serum globulin and heat-treated albumin, although blood derivatives, do not transmit the disease.

6. **Incubation period**—From 2 to 6 months, usually 12 to 14 weeks.
7. **Period of communicability**—Virus is demonstrable in blood of experimentally inoculated volunteers long before symptoms. How long harbored after infection is unknown; blood donors have infected recipients in each of three successive years. Some persons are carriers without experiencing clinically recognized attack.
8. **Susceptibility and resistance**—Susceptibility high.
9. **Methods of control**—
 - A. *Preventive measures:*
 1. Limiting administration of whole blood, and particularly pooled blood serum or plasma, to patients or others with clear indication of therapeutic usefulness or necessity. Pooling increases the likelihood that blood products will contain virus. Use of blood substitutes free of virus, such as human albumin or dextran, where feasible. Donor recruitment programs should rigidly exclude persons with past history of hepatitis.
 2. Thorough heat sterilization of syringes and needles, and of stylets for finger puncture. A fresh sterile syringe and needle or stylet is essential for each patient lest traces of blood from previous use contaminate these instruments.
 3. Storage of blood derivatives for 6 months at temperatures of about 32° C (90° F) is surest method of destroying hepatitis virus. Ultraviolet irradiation is not reliable.
 - B. *Control of patient, contacts, and the immediate environment:*
 1. Report to local health authority: Official report is advisable though not required in many states and countries, Class 3B (pp. 5, 6).
 2. Isolation: None; not known to be communicable except by injection.
 3. Concurrent disinfection: Of equipment contaminated with blood.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None; evidence on the usefulness of immune serum globulin is conflicting.
 7. Investigation of contacts and source of infection: Search for groupings of cases among others who have attended in common a clinic or hospital where parenteral therapy is much employed, such as a clinic for diabetes or syphilis.
 8. Specific treatment: None.
 - C. *Epidemic measures:* Surveys of persons receiving blood or blood products to determine incidence and to control technics associated with parenteral injections.
 - D. *International measures:* None.

HERPANGINA

1. **Identification**—An acute infection characterized by sudden onset with fever and small vesicular lesions of the pharynx which promptly ulcerate and cause moderate discomfort. The illness lasts 3 to 5 days, with occasional recurrence of fever 1 week later; no deaths. Definitive diagnosis during acute illness is by recovery of the in-

fectious agent from lesions, from stool specimens well into convalescence, and by response of neutralizing and complement-fixing antibodies.

Clinically, herpangina is to be distinguished from herpetic stomatitis which has larger, deeper, and more painful lesions commonly located in the front of the mouth.

2. **Occurrence**—Throughout the world, both sporadically and in epidemics, with greatest incidence in summer and early autumn. A common communicable disease of children under 10 years, but adult cases are relatively frequent.
3. **Infectious agents**—At least 6 distinct immunologic types of Coxsackie virus, Group A.
4. **Reservoir and source of infection**—Man is the reservoir. Sources of infection are pharyngeal (nose and throat) discharges and feces of infected persons, frequently in the absence of clinically recognized attack.
5. **Mode of transmission**—Direct contact with infected persons, and by droplet spread. Contaminated flies have been found. No reliable evidence of spread by insects, water, food, or sewage.
6. **Incubation period**—Usually 3 to 5 days.
7. **Period of communicability**—During the acute stage of illness and perhaps longer, since the virus persists in stools for several weeks.
8. **Susceptibility and resistance**—Susceptibility to infection is general. Immunity is acquired by infection, both clinical and inapparent; duration unknown. Second attacks occur with Group A Coxsackie virus of different immunological type.
9. **Methods of control**—
 - A. *Preventive measures*: None.
 - B. *Control of patient, contacts, and the immediate environment*:
 1. Report to local health authority: Obligatory report of epidemics; no case report, Class 4 (p. 6).
 2. Isolation: None.
 3. Concurrent disinfection: Of nose and throat discharges, feces, and articles soiled therewith.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Of no practical value.
 8. Specific treatment: None.
 - C. *Epidemic measures*:
 1. General notice to physicians of the prevalence or increased incidence of the disease, together with a description of onset and clinical characteristics.
 2. Isolation in bed of all children with fever, pending diagnosis.
 - D. *International measures*: None.

HISTOPLASMOSIS

1. **Identification**—A systemic mycosis of varying severity involving the reticuloendothelial system, primary lesion usually (if not always) in the lungs.

- A. *Asymptomatic*: Infection may be entirely asymptomatic and detectable only by acquired hypersensitivity to histoplasmin. No sequelae except that calcium sometimes is deposited in the primary lung lesion.
- B. *Acute benign respiratory*: Varies in severity from a mild respiratory illness to a temporarily incapacitating disease with general malaise, weakness, fever, chest pains, dry or productive cough, and eventual recovery with or without calcification of pulmonary and splenic lesions. This generalized infection approaches the progressive form of the disease, with varying degrees of hepato-splenomegaly and generalized lymphadenopathy, but with slow, spontaneous recovery.
- C. *Progressive fatal histoplasmosis*: Most frequent in infancy, occurs at all ages, and increases slightly at older ages. May be characterized by chronic cavitary lesions, hepato-splenomegaly, generalized lymphadenopathy, ulcers of the buccal mucosa, larynx, pharynx and intestines, and papular to pustular lesions of skin.

Clinical diagnosis is confirmed by demonstration of the fungus in smears and culture of sputum, exudates from ulcers, blood, or bone marrow. A rise in titer of complement-fixing antibodies supports the diagnosis, but antigens of *Histoplasma* and other pathogenic fungi are not specific and cross-reactions are sometimes strong.

Exposure to pigeon droppings should raise suspicion of cryptococcosis.

- 2. **Occurrence**—Reported from widely scattered areas in North, Central and South America, Europe, Africa, Hawaii, Indonesia, Japan, and the Philippines (130 countries). The severe disease occurs at all ages but more commonly in infants and in adults over 40 years; males outnumber females, especially at ages over 10 years where the ratio is 7 to 1. Histoplasmin hypersensitivity, indicating antecedent infection, is highly prevalent in eastern and central United States, infrequent in Rocky Mountain areas. The proportion of positive reactors increases from childhood to 30 years of age; differences by race and sex are not observed. Outbreaks of few to many cases have occurred in families or in groups of workmen with common exposure to environmental air-borne spores. Histoplasmosis occurs also in dogs, cats, rats, skunks, opossums, foxes, and other animals.
- 3. **Infectious agents**—*Histoplasma capsulatum*, a dimorphic fungus growing as a mold in soil where it produces conidia and macroconidia, and as a yeast in cells of the reticuloendothelial system. *H. duboisii* has been isolated from patients in Africa.
- 4. **Reservoir and source of infection**—Reservoir is saprophytic *Histoplasma* in soil around old chicken houses, in caves, in soil contaminated by bat droppings around houses sheltering the common brown bat, and in many miscellaneous soils with high organic content. Source of infection is soil or dust.
- 5. **Mode of transmission**—Through inhalation of air-borne spores of saprophytic origin; rarely by ingestion of spores contaminating food.
- 6. **Incubation period**—In reported epidemics, symptoms appear within 5 to 18 days after exposure, commonly 10 days.
- 7. **Period of communicability**—Under natural conditions not directly transmissible from man to man.
- 8. **Susceptibility and resistance**—Susceptibility is general. Inapparent infections are extremely common in endemic areas with probable resulting immunity except to heavy exposure. Clinical disease is regularly accompanied by circulating antibodies.

9. Methods of control—

- A. *Preventive measures:* Unavoidable exposure to contaminated environment may be minimized by spraying with water or disinfectant; masks may be worn.
- B. *Control of patient, contacts, and the immediate environment:*
 1. Report to local health authority: In selected endemic areas (USA); in many states and countries not a reportable disease, Class 3B (pp. 5, 6).
 2. Isolation: None.
 3. Concurrent disinfection: Discharges from skin lesions, sputum, and articles soiled therewith.
 4. Terminal disinfection: Thorough cleaning.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Household contacts of patients with systemic disease for evidence of infection from a common environmental source.
 8. Specific treatment: Amphotericin B appears to be the only satisfactory therapeutic agent now available; side effects require caution in its use.
- C. *Epidemic measures:* The occurrence of grouped cases of acute pulmonary infection in an endemic area, particularly with a history of exposure to dust within a closed space, should arouse suspicion of primary histoplasmosis. Suspected places such as chicken houses, barns, silos, caves, and basements should be investigated.
- D. *International measures:* None.

INFLUENZA

1. **Identification**—An acute highly communicable disease characterized by abrupt onset with fever of 1 to 6 days duration, chilliness or chills, discomfort, aches or pains in the back, legs or shoulders, and malaise or prostration. Respiratory symptoms of coryza, sore throat, or non-productive cough are a lesser feature of early influenza than the constitutional symptoms; usually a self-limited disease with recovery in 48 to 72 hours. Recognition is ordinarily on the basis of symptoms and the presence of an epidemic; sporadic cases are difficult to identify. Epidemic influenza derives its importance from the swiftness with which it strikes, the rapidity with which an epidemic evolves to threaten the normal functioning of a community, and from the complications that follow, especially pneumonia. Deaths are concentrated among the old, especially those debilitated by chronic cardiac, respiratory or metabolic disease, among women in late pregnancy, among infants, or whenever the acute illness is neglected.

Laboratory confirmation is by recovery of virus from throat washings or by demonstration of a significant rise in antibodies against influenza virus in serums obtained during acute and convalescent stages of the disease.

2. **Occurrence**—Variable; in pandemics, local epidemics, and as sporadic cases, the latter commonly unrecognized. Epidemics, the most usual pattern of recognized occurrence, may affect up to half a population

within 4 to 6 weeks; attack rates ordinarily vary from less than 5% to 20 or 30%. In temperate zones epidemics tend to occur in winter, in the tropics irregularly. The disease shows a cyclic tendency, with influenza A appearing in epidemic form at shorter intervals than influenza B. A single serologic type of virus is usually present among patients of the same epidemic, but presence of 2 types is known. Highly fatal pandemics of influenza occurred in 1889 and in 1918; subsequent outbreaks have been milder. The 1957-1958 pandemic of Asian influenza was clinically mild but extremely widespread.

3. **Infectious agent**—Two types of influenza virus, Influenza A and Influenza B, are long recognized; Influenza C, more recently identified, has thus far appeared only in localized outbreaks. Types A and B include numerous serologically distinct strains. Three families of type A virus have been isolated from man,—A (PR8 1934), A1 (FM1 1947), and A2 (1957 Asian strain); other viruses possessing the common type A complement fixing antigen, have been isolated from animals including swine, horses, duck, and fowl. There is serological evidence that a virus closely related to the swine virus was present in man about the time of the 1918 pandemic. At least 2 families of type B virus are recognized; type C appears to be antigenically homogenous. Within periods as short as 10 to 15 years the family of strains once prevailing tends to be replaced by others of somewhat different antigenic nature. Continued epidemiologic and serologic studies are therefore necessary to guide preventive measures.
4. **Reservoir and source of infection**—Man is the reservoir. Sources of infection are discharges from the mouth and nose of infected persons.
5. **Mode of transmission**—By direct contact, through droplet infection, or by articles freshly soiled with discharges of the nose and throat of infected persons; possibly air-borne.
6. **Incubation period**—Short, usually 24 to 72 hours.
7. **Period of communicability**—Probably limited to a brief period before clinical onset and 1 week thereafter.
8. **Susceptibility and resistance**—Susceptibility is greatest in childhood, with the disease most frequent at about 10 years of age. Incidence by age tends to decline progressively thereafter. The presence of serum antibody in unvaccinated persons more than 3 months old is to be interpreted as the result of infection. The antibody spectrum broadens with age because of repeated exposure to various strains and different races of influenza virus; immunity at a prescribed time thus depends on the composite of antibodies against principal antigens of influenza virus, as determined by age of the person and opportunities for exposure afforded by the particular environment. The antibody content of different age segments of a population for different types of virus and for different antigenic families of strains within a type, reflects the prior experience of that population.

Immunity after attack against homologous or closely related strains may persist for several years but freedom from disease is often short because of the variety of influenza viruses. The many other microbial agents capable of inducing an influenza-like syndrome confuse the clinical evaluation of duration of immunity.

9. Methods of control—

A. Preventive measures:

1. Active immunization is the only known method of achieving a substantial reduction in incidence of influenza. Vaccines are effective

when the antigenic component of the vaccine matches rather closely the prevailing strain of virus.

Routine inoculation of whole general populations is not feasible. Special consideration should be given protection of those at unusual risk of complications or of death (see Section I, Identification), and those engaged in essential community services. Where indicated, immunization should be repeated at yearly intervals well before the expected seasonal occurrence. Major shifts in antigenic structure of influenza virus may reduce the effectiveness of immunization.

2. Education of the public as to the sanitary hazards arising from spitting, sneezing, and coughing in the close presence of others, and the inherent risk in common use of towels, drinking glasses, eating utensils, or toilet articles. Promote the use of disposable paper handkerchiefs.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory report of epidemics resembling influenza; no individual case report, Class 4 (p. 6). Confirmation of the epidemic disease as influenza should be sought promptly through laboratory procedures.
2. Isolation: During the acute illness, not as an official requirement but as directed by the attending physician, and primarily for the protection of the patient.
3. Concurrent disinfection: Discharges from the nose and throat of the patient.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None; too late to be effective.
7. Investigation of contacts and source of infection: Of no practical value.
8. Specific treatment: None. Sulfonamides and antibiotics do not affect the uncomplicated disease; they should be employed only if pulmonary complications arise.

C. Epidemic measures:

1. Inoculation with vaccine after exposure is useless. Vaccination of the community after introduction of the virus is of little value because the disease spreads rapidly and protection does not follow for a week or longer. For practical effect, persons must be immunized in advance of an anticipated epidemic; international control measures under 9D2 and 9D3 are designed to make this possible; the same procedures are applicable within a country.
2. To minimize severity and to protect the patient from secondary infections, patients should go to bed at the beginning of an attack and not return to work until fully recovered, preferably with the approval of a physician.
3. The unnecessary aggregation of large numbers of persons is to be discouraged.
4. Unnecessary hospitalization should be discouraged, thereby avoiding the crowding of beds in hospitals and institutions to accommodate increased numbers of patients or inmates; in fact, increased spacing between beds in wards and in dormitories reduces the risk of attack and of pneumonia.

5. Continued provision to local health authorities, by state and national health agencies, of current information on the extent and nature of the existing epidemic.

D. International measures:

1. Prompt notification to WHO of epidemics within a country, especially those involving institutional, military, or other considerable groups.
2. Identification of virus type in such situations through prompt dispatch of the necessary blood samples or throat washings to one of the WHO influenza centers which now number close to 60, and are widely distributed in many countries. The World Influenza Center in London and the International Influenza Center for the Americas, Atlanta, Ga., serve as reference centers for recognition and comparison of new or unusual virus types.
3. Continuing epidemiologic studies and exchange of information by national health agencies, between countries and with WHO, are essential to establish broad movements of epidemic influenza, to facilitate early recognition of outbreaks within a country, and to identify the prevailing type and strain of virus.
4. Continuing attempts to mobilize development and production facilities to permit rapid shipment and use of an appropriate vaccine when a new strain appears and threatens to become pandemic.

KERATOCONJUNCTIVITIS, EPIDEMIC

1. **Identification**—An acute infection of the eye with unilateral or bilateral inflammation of conjunctivae, and edema of lids and periorbital tissues, often with serous or seromucoid exudate. Onset is sudden, with low-grade fever and often headache and malaise. Tender preauricular lymphadenopathy usually occurs within 2 to 4 days. Opacities of the cornea, resulting from subepithelial infiltrates which commonly do not ulcerate, are evident within 4 to 14 days. Duration of illness is 2 to 4 weeks and complete recovery is usual although keratitis with impairment of vision may persist in 1 to 10% of patients. Synonym: Infectious Punctate Keratitis.
Clinical diagnosis is confirmed by isolation of the virus in tissue culture from conjunctival scrapings, or by demonstration of specific antibody response in acute and convalescent serums.
2. **Occurrence**—Presumably world-wide. Both sporadic cases and large outbreaks occur in the Far East, Hawaii, North America, and Europe. Outbreaks among industrial employees in temperate climates commonly involve only a small part of the population at risk.
3. **Infectious agent**—Type 8 adenovirus, occasionally other types.
4. **Reservoir and source of infection**—Man is the only known reservoir; source of infection is discharges from eye of an infected person.
5. **Mode of transmission**—Direct contact with discharges from eye of an infected person or with articles freshly soiled with conjunctival or nasal discharges of such person. In shipyards and industrial plants trauma to the conjunctivae from dust and dirt, and contamination of the eye in first-aid stations and dispensaries where proper aseptic technics are not employed, are recognized factors in transmission. A number of cases have originated in eye clinics and the offices of private physicians.

6. **Incubation period**—Not established for natural infections but 5 to 7 days is usual in experimental infection of volunteers.
7. **Period of communicability**—Indefinite, but certainly during acute stage of the disease.
8. **Susceptibility and resistance**—No age, sex, or race is known to be immune. Some degree of immunity follows an attack. Recurrent infections have been reported.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Education as to personal cleanliness and the danger in use of common towels and toilet articles.
 2. Use of safety measures such as goggles in industrial plants and ship-yards.
 3. Rigid asepsis in the medical divisions of factories and plants to prevent spread of infection by hands of attendants or by instruments, particularly in ophthalmological procedures.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6).
 2. Isolation: Advisable during the acute stage of the disease.
 3. Concurrent disinfection: Of conjunctival and nasal discharges and articles soiled therewith.
 4. Terminal disinfection: Airing and sunning of room and bedding.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: To locate other cases and institute precautions at home or place of work.
 8. Specific treatment: None.
 - C. **Epidemic measures:**
 1. Intensify educational efforts with respect to hygiene of the eye.
 2. Organize convenient facilities for prompt diagnosis and treatment.
 - D. **International measures:** None.

LARVA MIGRANS, VISCERAL

1. **Identification**—A chronic and usually mild infection with larvae of nematode parasites of the dog and cat. Marked eosinophilia, hepatomegaly, hyperglobulinemia, and fever are clinical manifestations of diagnostic importance. Symptoms may be present for as long as a year. Pneumonitis or neurological disturbances may occur; rarely fatal. Demonstration of larvae or the typical eosinophilic granulomatous lesions by liver biopsy is the only definitive method of confirming the diagnosis. Serodiagnostic tests are helpful but are not generally available. Aberrant larvae of human helminths, *Ascaris lumbricoides*, *Strongyloides stercoralis*, *Necator americanus*, and *Ancylostoma duodenale* may be found occasionally in tissues including some ectopic sites, but they do not persist and migrate in the tissues for long periods as do the larvae of *Toxocara*.

2. **Occurrence**—Probably world-wide, wherever dogs and cats harbor *Toxocara*. The disease has had most attention in the United States, but incidence is probably no greater than in most countries. Occurrence is occasional, as isolated cases in a family. Limited mainly to children aged 14 to 40 months; occurs uncommonly in mentally retarded children of older age. The next older or younger sibling often shows eosinophilia or other evidence of light or residual infection; no differences by sex. Epidemics are unknown.
3. **Infectious agents**—Larvae of *Toxocara canis* of the dog and *T. cati* of the cat.
4. **Reservoir and source of infection**—Reservoir is dog and cat. Source of infection is contaminated soil containing infective *Toxocara* eggs from canine and feline feces deposited about dooryards.
5. **Mode of transmission**—By direct or indirect transmission of embryonated eggs to the mouth from soil or other contaminated material; directly related to the practice by young children of eating dirt. Eggs require several weeks incubation to become infective. After ingestion the embryonated eggs of *Toxocara* hatch in the intestine, larvae penetrate the wall, and migrate to liver and lungs by lymphatic and circulatory systems. From the lungs the larvae are spread by the systemic circulation to various organs, causing damage by their wanderings and through tissue sensitization.
6. **Incubation period**—Probably weeks or months depending upon the intensity of infection, number of reinfections, and sensitivity state of the patient.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—Children 1 to 4 years of age most commonly have symptomatic infections. The apparently greater tolerance of older children and adults is probably related to lesser exposure. Dogs may acquire infection as puppies and remain infective for life; infection often ends on reaching sexual maturity. Adult male dogs are more frequently infected than females. Sex and age differences are less marked for cats; older animals somewhat less susceptible than young.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Prevention of soil contamination by the feces of dogs and cats in areas immediately adjacent to houses and play areas of children, especially in multiple housing projects.
 2. Stools of dogs and cats passed in play areas should be buried deeply, or otherwise disposed of. Children's sand boxes offer an attractive site for defecating cats; they should be covered when not in use.
 3. Dogs and cats under 6 months should be treated routinely for removal of worms and periodically thereafter as indicated. Worms and feces passed as a result of treatment should be destroyed.
 4. Education of the family as to source and origin of the infection, particularly the practice of eating dirt.
 5. Encouragement of children in the practice of hand washing after play in the soil and before eating.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).

2. Isolation: None.
 3. Concurrent disinfection: None.
 4. Terminal disinfection: Premises where infections have occurred can be disinfected by raw salt or borax. These agents destroy lawns or any vegetation; applicable particularly to beaches, playgrounds, and areas under houses.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Recognition of the disease in a patient often leads to discovery of infected siblings or playmates.
 8. Specific treatment: None.
- C. Epidemic measures: Not applicable.
- D. International measures: None.

LEISHMANIASIS, CUTANEOUS

1. Identification—Localized superficial infection of reticuloendothelial macrophages by flagellate protozoa of the genus *Leishmania*, producing nodular and ulcerating indolent lesions of any exposed part of the body. The lesion is the result of a bite by the infected vector, except for secondary lesions produced by scratching. In some parts of tropical America and Africa the lesions are more ulcerative, and often involve mucous membranes, causing extensive necrosis of nose, mouth, and pharynx; secondary infection and complications may cause death. Synonyms: In Old World: Aleppo, Baghdad or Delhi Bait, Oriental Sore; in New World: Espundia, Uta, Chiclero or Bauru Ulcer.

Definitive diagnosis is by microscopic identification of *Leishmania* bodies in stained smears of scrapings from edges of lesions; also by culture on suitable media of material aspirated from lesions. The intradermal test, using an extract or suspension of dead leptomonads, may aid; no serological test of practical value.

2. Occurrence—Old World: Northwest India, West Pakistan, the Middle East, southern Russia, the Mediterranean littoral, North, West, and Central Africa. New World: endemic in Mexico (especially Yucatan), most of Central America, and every country of South America except Chile. Occasionally seen in the United States in persons coming from endemic areas.
3. Infectious agents—*Leishmania tropica* (Old World). *Leishmania brasiliensis* (New World).
4. Reservoir and source of infection—Reservoirs are infected persons with exposed lesions containing parasites, also dogs, cats, jackals, and gerbils. The spiny rat has been found naturally infected in Panama. Sandflies of the genus *Phlebotomus* are the immediate source of infection.
5. Mode of transmission—Commonly through bite of infected sandflies, *Phlebotomus*, possibly by direct contact of abraded skin with lesion of another person; perhaps mechanically by other flies, but doubtful.
6. Incubation period—From a few days to many months.
7. Period of communicability—As long as parasites remain in lesions; in untreated cases this may be for a year or longer. Spontaneous heal-

ing is the rule except with destructive lesions of mucous membrane. Duration of infectivity of vector unknown.

8. **Susceptibility and resistance**—Susceptibility is general. Immunity is usual after an infection that has healed spontaneously.

9. **Methods of control**—

A. **Preventive measures:**

1. Periodic application of insecticides with residual action to exterior and interior of doorways and all other openings in habitations through which *Phlebotomus* can enter. This insect flies in small "hops," spends much time on surfaces, and thus is especially vulnerable to contact insecticides. Possible breeding places should be sprayed, such as rubbish heaps, stone walls, duck and chicken pens, animal houses, and similar damp situations near dwellings.
2. Residual spraying may be supplemented by aerosol spraying of bed nets, and use of insect repellents.
3. Sanitary housing, providing adequate light and ventilation, and eliminating damp floors, thatched roofs, rubbish piles, vegetation near house, and animals stabled in the house.
4. Since *Phlebotomus* does not fly far, dwellings in infected areas should be located not less than 150 meters from the forest. Avoid entrance into infected areas after sundown.
5. Wherever dogs, rodents, or other mammals serve as natural reservoirs their destruction will reduce the incidence of disease in the human population.
6. Education of the population concerning the mode of transmission and methods of controlling *Phlebotomus*.
7. Vaccination using scrapings from active lesions of oriental sore has been practiced successfully by native peoples. Active immunization with a live vaccine of *L. tropica* prepared from cultures or infected chick embryo, provides protection against oriental sore. Immunity develops after several months. Vaccination with dead *Leishmania* has been tried against *L. braziliensis* with satisfactory results.

B. **Control of patient, contacts, and the immediate environment:**

1. Report to local health authority: In selected endemic areas; in many countries not a reportable disease, Class 3B (pp. 5, 6).
2. Isolation: None; protect patient from bites of *Phlebotomus* by fine mesh screens and mosquito nets (45 to the linear inch), by spraying of quarters with insecticides having residual action and by the use of repellents.
3. Concurrent disinfection: None, other than destruction of sandflies in dwelling.
4. Terminal disinfection: None, other than destruction of sandflies in dwelling.
5. Quarantine: None.
6. Immunization of contacts: Active immunization for *L. tropica* infection, oriental sore.
7. Investigation of contacts and source of infection: Search for other cases among contacts and for *Phlebotomus*; also breeding areas of sandflies around dwellings.
8. Specific treatment: Pentavalent compounds of antimony as for kala azar. Secondary bacterial infection is treated with appropriate systemic or local antibiotics or by chemotherapy.

C. *Epidemic measures*: In areas of high incidence intensive efforts to control the disease by provision of diagnostic facilities, by mass treatment campaigns and by measures against sandflies.

D. *International measures*: None.

LEISHMANIASIS, VISCERAL

1. **Identification**—An infection of the reticuloendothelial system caused by a flagellate protozoan of the genus *Leishmania* and characterized by irregular fever of long duration, chronicity, hepatosplenomegaly, lymphadenopathy, anemia with leucopenia, and progressive emaciation and weakness. Untreated, a highly fatal disease. Fever is of gradual or sudden onset, often with two daily peaks; alternating periods of apyrexia and low grade fever follow. Common complications are pneumonia, gangrenous stomatitis, dysentery. After chemotherapy cutaneous lesions containing *Leishmania* may occur. Synonym: Kala Azar.

Diagnosis is by finding Leishman-Donovan bodies in stained smears from bone marrow, spleen, liver, lymph node, or blood (which requires careful examination); by culture of such materials, or by injection into hamsters. Reversal of albumin-globulin ratio and complement-fixation are presumptive tests.

2. **Occurrence**—A rural disease. Endemic in Asia; in eastern India: States of Assam, Bengal, Bihar, Madras; in east Pakistan and in China, chiefly north of the Yangtze River as far as south Manchuria; in the Middle East: Turkey, Syria, Lebanon, Israel, Jordan, Yemen, and Saudi Arabia. In Europe: southern Russia, the Caspian littoral, Portugal, and the Mediterranean islands; in Africa: Morocco, Tunis, Algeria, Tripolitania, Egypt, Sudan, Ethiopia, Somaliland, northern Kenya, French Equatorial Africa, Ghana, and Nigeria. In South America: northern Argentina, Paraguay, northeastern Brazil, Venezuela and Colombia; also El Salvador, Guatemala, and southern Mexico.

3. **Etiologic agent**—*Leishmania donovani* (L. infantum).

4. **Reservoir and source of infection**—Reservoir is usually man, an infected person with parasites in reticuloendothelial cells or blood. The high incidence of infection among dogs and cats in some endemic areas suggests these animals as reservoirs of human infection; jackals and gerbils also harbor the infection. The immediate source of infection is *Phlebotomus*, a sandfly.

5. **Mode of transmission**—Through bite of infected sandflies of the genus *Phlebotomus*. The fly is infected by sucking the peripheral blood or by ingesting parasites present in skin of an infected individual.

6. **Incubation period**—Generally 2 to 4 months; as short as 10 days or as long as 24 months.

7. **Period of communicability**—As long as parasites persist in circulating blood or skin, sometimes after treatment and clinical recovery from visceral infection. Duration of infectivity of sandfly is unknown.

8. **Susceptibility and resistance**—Susceptibility is general. In the Mediterranean area most cases occur in children under 5 years of age. In other endemic areas peak incidence is at 5 to 14 years, with many cases in adults. Clinical recovery apparently leads to lasting immunity. Recovery from cutaneous leishmaniasis does not confer immunity against kala azar or vice versa.

9. Methods of control—

A. Preventive measures:

1. The control of sandflies. The measures are those listed under Leishmaniasis, Cutaneous 9A1-6, p. 101.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in many countries not a reportable disease, Class 3B (pp. 5, 6).
2. Isolation: None. Protect patient from bites of *Phlebotomus* by fine mesh screens and mosquito nets, by spraying quarters with insecticide having residual action, and by use of repellents.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization: None.
7. Investigation of contacts and source of infection: Search for sandflies on the immediate premises or for other contact with them.
8. Specific treatment: The pentavalent compounds of antimony (neostibosan, solustibosan, and urea stibamine) are effective. Antimony-resistant and antimony-sensitive cases may be treated by the diadimidine compounds (stilbamidine, pentamidine), not used routinely because of their toxicity. In Mediterranean areas and the Sudan the parasite is more resistant than in India and requires more energetic treatment.

C. Epidemic measures:

1. Surveys in areas of high incidence and location of endemic foci.
2. Intensive efforts to eliminate the disease by mass treatment campaigns and by the control of sandflies.

- D. *International measures:* Coordinated programs of control entered into by neighboring countries where the disease is endemic.

LEPROSY

1. **Identification**—A chronic communicable disease characterized by lesions of the skin—infiltration, macules, plaques, papules, and nodules—and by involvement of peripheral nerves with consequent anesthesia, muscle weakness and paralysis, and trophic changes in skin, muscle, and bone. In lepromatous leprosy, the mucous membranes of the upper respiratory tract are also usually invaded. Progress of the disease is slow; death is usually due to other causes.

Demonstration of acid-fast bacilli in suspected lesions is confirmatory in lepromatous cases; bacilli may be rare or not demonstrable in tuberculoid (maculo-anesthetic) lesions and diagnosis depends primarily upon presence of anesthesia in areas served by affected nerves. The leprosy bacillus can not be cultured and is nonpathogenic for animals other than man.

2. **Occurrence**—Mostly in the tropics and subtropics. Prevalence rates of 5 per 1,000 or higher are found only in the tropics, the highest reported rates being in French Equatorial Africa, French West Africa, and the Belgian Congo. A few countries with temperate climates have estimated rates of 1 per 1,000, including China, Japan, and Korea. China and

India have about one-half of the estimated world total of 3 to 4 million cases. In Europe, low endemicity in Greece, Portugal, Spain; residual foci only in several other countries. Endemic but decreasing in Hawaii; present at a low level in Canal Zone, Puerto Rico, and Virgin Islands. Southeastern Texas is the chief focus in continental United States, producing about 20 indigenous cases annually. Occasional cases occur in Louisiana and rarely in Florida (Key West area) and in Southern California.

3. **Infectious agent**—*Mycobacterium leprae*, leprosy bacillus.
4. **Reservoir and source of infection**—Reservoir is man. Source of infection is discharges from lesions of infected persons.
5. **Mode of transmission**—Not definitely known; the bacillus probably gains entrance through skin or mucous membrane of the upper respiratory tract.
6. **Incubation period**—Prolonged, undetermined, from a few months to several years.
7. **Period of communicability**—Commences when lesions become open and discharge leprosy bacilli; continues until healing, communicability being shortened by adequate chemotherapy. Patients with demonstrable acid-fast bacilli in smears from skin or mucous membrane are potentially open cases even if demonstrable ulceration is not present.
8. **Susceptibility and resistance**—No racial immunity. May be contracted in adult life but is more frequent in persons exposed in infancy and early childhood. Natural reactivity to lepromin may indicate some degree of resistance; it is absent in the lepromatous (more malignant) type, usually present in the tuberculoid, and frequently present in healthy persons.
9. **Methods of control**—

A. Preventive measures:

1. Infants should be separated from leprous parents at birth; educational efforts should stress greater risk when exposure is early in life. The exaggerated fear of leprosy and belief in its high communicability should be discounted.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report obligatory in most states and countries and desirable in all, Class 2B (p. 5).
2. Isolation: In endemic areas patients with demonstrated leprosy bacilli in smears from lesions should be isolated in hospitals or colonies and treated until bacteriologically negative for at least 6 months. The next best procedure is institutional treatment until ulcerative lesions are healed, followed by home isolation under medical supervision. In many countries of high endemicity only supervision through clinics is practicable. In areas where the disease is not indigenous, home isolation is sufficient. Patients discharged from institutions should be examined periodically, the suggested interval being 6 months.
3. Concurrent disinfection: Discharges of lesions and articles soaked therewith.
4. Terminal disinfection: Thorough cleaning of premises where patient has lived.
5. Quarantine: None.

6. Immunization of contacts: None. Vaccination with BCG will induce reactivity to lepromin.
7. Investigation of contacts and sources of infection: Should be undertaken in cases of apparently recent origin; the long and uncertain period between exposure and recognition makes discovery of the source of infection difficult. Periodic examination of contacts to determine secondary cases.
8. Specific treatment: Sulfones (promin, diasone and DDS, diamino-diphenyl-sulfone), the preferred therapeutic agents, are continued for long periods. Promin is administered intravenously, diasone orally, with slowly increased doses. Initial small oral doses of DDS are slowly increased to tolerance. Streptomycin probably has value equal to sulfones and may be substituted when sulfone therapy fails or is not tolerated. Isoniazid is not effective. A number of other drugs are under evaluation. Penicillin is of value only for control of secondary infection.

C. Epidemic measures: In areas of high endemicity or in those uncommon situations justifying epidemic characterization, the program of control includes provision of facilities for bacteriological diagnosis, treatment clinics, case finding to include examination of family contacts of known cases every 6 months, separation of children of leprous parents from birth, also the usual measures of reporting and isolation.

D. International measures:

1. Exercise of the recognized international rights of governments to refuse entry of immigrants who are found to have leprosy is usual and desirable.
2. Reciprocal measures between governments at authorized points of entry to prevent introduction or spread of the disease by immigrants.

LEPTOSPIROSIS

1. Identification—A group of acute systemic infections characterized by fever, headache, chills, severe malaise, vomiting, muscular aches, meningeal irritation, and conjunctivitis; infrequently jaundice, renal insufficiency, hemolytic anemia, and hemorrhage in skin and mucous membranes. Pretibial rash occurs occasionally. Leucocytosis is common, pleocytosis of spinal fluid frequent. The acute illness lasts from 1 to 3 weeks; relapses may occur. Fatality is low but increases with advancing age; may reach 20% or more in severe cases with jaundice and kidney damage, formerly considered the common manifestations of the disease. Synonyms: Weil's Disease, Canicola Fever, Swineherd Disease, Mud Fever, Hemorrhagic Jaundice, Fort Bragg Fever, Rice Field Fever and Cane Field Fever.

Agglutination tests serve usefully in confirmation of clinical diagnosis; also complement-fixation tests.

2. Occurrence—Outbreaks among swimmers exposed to contaminated waters are reported. An occupational hazard to rice field workers, farmers, sewer workers, miners, veterinarians, animal husbandmen, abattoir workers, fish workers, and those who live or work in rat-infested premises. Distribution of reservoirs of infection and of one or another species of *Leptospira* is probably world-wide.

3. **Infectious agents**—Many species of the genus *Leptospira*, *L. icterohemorrhagiae*, *L. canicola*, *L. autumnalis*, and *L. pomona*, have been recovered from human cases in the United States; others are probable. At present at least 21 serogroups containing 59 serotypes are recognized.
4. **Reservoir and source of infection**—Reservoirs include cattle, dogs, and swine. Rats and other rodents are frequently infected as are foxes, skunks, raccoons, and opossums. Water birds have been reported as carriers. The source of infection is urine of infected animals and possibly infected tissues.
5. **Mode of transmission**—Contact with water contaminated with urine of infected animals, as in swimming or accidental or occupational immersion; direct contact with infected animals. Infection presumably results from penetration of abraded skin or mucous membrane, or possibly through ingestion.
6. **Incubation period**—Four to 19 days, usually 10 days.
7. **Period of communicability**—Under natural conditions direct transmission from man to man is negligible.
8. **Susceptibility and resistance**—Susceptibility of man is general.
9. **Methods of control**—

A. Preventive measures:

1. Protection with boots and gloves of workers exposed to hazard of infection.
2. Avoidance of swimming or wading in potentially contaminated waters.
3. Rodent control in human habitations, particularly rural and recreational.
4. Segregation of domestic animals, and prevention of contamination of living and working areas of man by urine of infected animals.
5. Vaccines have been used to a limited extent. It is essential that they represent the dominant local strain.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory case report in many states and countries, Class 2B (p. 5).
2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Search for exposure to infected animals or history of swimming in contaminated waters.
8. Specific treatment: Penicillin, streptomycin, and tetracycline antibiotics are leptospiricidal *in vitro*, but not yet of demonstrated value in treatment of human infections.

C. Epidemic measures: Search for source of infection, as a pond used for swimming; eliminate contamination or prohibit further use. Investigate possible industrial or occupational sources of infection.

D. International measures: None. Seven leptospirosis reference laboratories operate throughout the world under WHO auspices.

LOAIASIS

1. Identification—A nematode infection characterized by migration of the adult worm through the subcutaneous or deeper tissues of various parts of the body causing transient "fugitive swelling" or "Calabar swelling" of the trunk and extremities. Migration through the bulbar conjunctivae is attended by marked pain and edema. A chronic and relatively benign disease.

Female worms produce larvae, microfilariae, which have a diurnal periodicity. They are best demonstrated in stained thick blood smears or stained sediment of laked blood. If microfilariae are not demonstrable in the blood, the intradermal test using *Dirofilaria* antigen is diagnostically valuable, although nonspecific.

Infection with other filaria such as *W. bancrofti* (p. 78) and *A. perstans* requires differentiation in endemic areas.

2. Occurrence—Loiasis is widely distributed in tropical West and Central Africa. In the Belgian Congo up to 90% of indigenous inhabitants of some villages are infected. Newcomers to these areas are commonly infected.

3. Infectious agent—*Loa loa*, a nematode worm.

4. Reservoir and source of infection—Reservoir is an infected person harboring microfilariae in the blood. Source of infection is the mango fly of the genus *Chrysops*.

5. Mode of transmission—*Chrysops dimidiata*, *C. silacea* and other species ingest blood and microfilariae. The larvae develop in the muscles and connective tissues of the fly. The developed filariform larva migrates to the proboscis and is transferred to the next human host by bite of the infected fly. Development in the fly takes from 10 to 20 days.

6. Incubation period—Symptoms usually do not appear until several years after infection but may occur as early as four months. Microfilariae do not appear in the peripheral blood until a year or more.

7. Period of communicability—The adult worm may live in man as long as 17 years and microfilariae may be present in the blood during this time; in the fly, communicability is from 10 to 20 days after its infection until all infective larvae have migrated from it or until death of the fly.

8. Susceptibility and resistance—Susceptibility is universal; repeated infections occur and immunity if present has not been demonstrated.

9. Methods of control—

A. Preventive measures:

1. Measures directed against the aquatic larvae have not proved practical or effective, as breeding areas are usually extensive.
2. Dimethylphthalate applied to the exposed skin is an effective vector repellent.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health department: In most countries not a reportable disease, Class 3C (pp. 5, 6).
2. Isolation: Not practicable. So far as possible patients with microfilariae in the blood should be protected from *Chrysops* bites as a means of reducing transmission.
3. Concurrent disinfection: None.

4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: None; a community problem.
 8. Specific treatment: Diethylcarbamazine (hetrazan®) causes disappearance of microfilariae, reduces frequency and intensity of Calabar swellings, and may kill adult worm with resulting cure.
- C. *Epidemic measures*: In hyperendemic areas vector control is practicable only if aquatic breeding areas are of limited extent.
- D. *International measures*: None.

LYMPHOCYTIC CHORIOMENINGITIS

1. **Identification**—An endemic viral infection of animals, especially mice, transmissible to man with a marked diversity of clinical manifestations; may begin with an influenza-like attack and terminate by recovery, or after a few days of more or less complete remission, meningeal symptoms suddenly appear. Attack is sometimes initiated by meningeal symptoms. Patients with severe meningoencephalitis have somnolence, disturbed deep reflexes, paralysis, and anesthesia of skin. Course is usually short and most patients recover within a few weeks; occasionally fatal. Spinal fluid, sterile to bacterial culture, has from a few hundred to more than 5,500 cells per cmm, almost wholly lymphocytes.

Laboratory diagnostic methods include isolation of virus from blood, urine, nasopharynx or spinal fluid early in attack; inoculation of guinea pigs intraperitoneally or of mice intracerebrally; or comparison of titers of neutralizing or complement fixing antibodies in serums collected early and late in the disease, an interval of 6 weeks often being necessary.

The diseases included under the Aseptic Meningitis Syndrome (p. 59) require differentiation, as do a number of arthropod-borne viral infections (p. 27).

2. **Occurrence**—Uncommon, but not to the extent that numbers of recognized cases indicate. Foci of infection often persist within limits of a city block for months or years, giving sporadic clinical disease.
3. **Infectious agent**—The virus of lymphocytic choriomeningitis.
4. **Reservoir and source of infection**—Reservoir is the infected house mouse, *Mus musculus musculus*; naturally infected guinea pigs, monkeys, dogs, and swine have been observed. Sources of infection are urine and feces of mice.
5. **Mode of transmission**—Transmission to man is probably through contaminated food or dust, possibly by arthropods.
6. **Incubation period**—Information is limited; probably 8 to 13 days from infection to systemic manifestations, 15 to 21 days to meningeal symptoms.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Naturally infected mice may carry the virus through life; the infected female transmits virus to offspring.
8. **Susceptibility and resistance**—Unknown. Virus neutralization by blood serum of persons recovered from the disease, it also occurs commonly in persons without history of recognized attack.

9. Methods of control—**A. Preventive measures:**

1. General cleanliness of home and place of work, with elimination of mice.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
2. Isolation: None.
3. Concurrent disinfection: Of discharges from the nose and throat, of urine and feces, and of articles soiled therewith.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Home and place of employment for presence of house mice, with attention also to arthropods.
8. Specific treatment: None.

C. Epidemic measures: Not applicable.**D. International measures:** None.

LYMPHOGRANULOMA VENEREUM

1. **Identification**—A venereally acquired virus infection of lymph channels and lymph nodes manifesting itself in a variety of ways: bubo formation, ulceration, elephantiasis of genitalia, and rectal stricture. The disease sometimes begins with a small painless evanescent erosion, papule, or herpetiform lesion, followed shortly by acute, subacute or chronic adenitis and periadenitis, usually with multiple foci of suppuration and with grooved adherent skin of a purplish hue; more commonly the bubo is the first manifestation. Constitutional symptoms during lymphatic progression include fever, chills, headache, vague abdominal aches, joint pains, and anorexia. Spontaneous regression of buboes does not indicate recovery; the course is often long, disability is great, but the disease is essentially nonfatal. Synonyms: Lymphogranuloma Inguinale, Climatic Bubo, and Lymphopathia Venereum.

Diagnosis is aided by skin test with Frei antigen or by demonstration of complement-fixing antibodies against lymphogranuloma venereum-psittacosis group of viruses; neither test is conclusive.

2. **Occurrence**—A commoner disease than ordinarily believed; widespread throughout the world, especially in tropical and subtropical areas. Endemic in southern United States, particularly among the lower social classes. Age incidence is that of greatest sexual activity, with the disease most frequent among the sexually promiscuous. Sex differences are not pronounced and all races are affected.
3. **Infectious agent**—The virus of lymphogranuloma venereum, immunologically related to the virus of psittacosis.
4. **Reservoir and source of infection**—Reservoir is man. Sources of infection are lesions of rectum and urethra and sinuses and ulcerations of infected persons.

5. **Mode of transmission**—Through direct contact during sexual intercourse; by indirect contact with articles contaminated by infectious exudates, especially infections acquired by children.
6. **Incubation period**—Five to 21 days to primary lesion, usually 7 to 12; if inguinal bubo is first manifestation, 10 to 30 days, sometimes several months.
7. **Period of communicability**—Variable, from weeks to years, during presence of active lesions.
8. **Susceptibility and resistance**—Susceptibility seemingly is general. Immunity does not follow attack.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Except for specific information relating to lymphogranuloma venereum, preventive measures are those for the venereal diseases (see Syphilis 9A, p. 187).
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: In selected endemic areas (USA, some states); in most states and countries not a reportable disease, Class 3C (pp. 5, 6).
 2. Isolation: None. Avoid sexual contact until lesions are healed.
 3. Concurrent disinfection: None; care in disposal of discharges from lesions and of articles soiled therewith.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: Not applicable; prompt treatment on recognition or clinical suspicion of infection.
 7. Investigation of contacts and source of infection: Search for sexual contacts exposed prior and subsequent to appearance of disease.
 8. Specific treatment: Varies with stage of the disease; sulfadiazine is the drug of choice in the bubo phase; tetracyclines may be used. For proctitis and other ulcerative lesions, tetracycline antibiotics administered orally for 10 days or longer as indicated by clinical response.
 - C. **Epidemic measures:** Intensification of preventive and control activities.
 - D. **International measures:** See Syphilis 9D, p. 188.

MALARIA

1. **Identification**—A systemic disease, acute, sometimes severe and often chronic, commonly beginning with indefinite malaise shortly followed by a characteristic shaking chill with rapidly rising temperature, usually accompanied by headache and nausea and ending with profuse sweating. After an interval free of fever, the cycle of chills, fever, and sweating is repeated. As the disease progresses the paroxysms tend to occur daily, every other day or every third day, depending on species of malaria parasite. Duration of untreated primary attack varies from a week to a month or longer. Relapses are common and may occur at irregular intervals for several years. Fatality in untreated cases varies from less than 1% to 10%, rarely higher, depending on character of the parasite and degree of host resistance. In treated cases the fatality

may be 0.1% and rarely exceeds 0.5%. Clinical diagnosis depends on characteristic intermittent chills and fever, enlargement of the spleen, and secondary anemia, often with mild icterus.

Laboratory confirmation should always be sought through demonstration of malaria parasites in blood films by microscopic examination. Repeated examinations may be necessary; the thick film method is most likely to reveal the parasite; often not demonstrable in films from patients recently or actively under treatment.

2. **Occurrence**—Endemic malaria essentially has disappeared from many countries where malaria was a health problem a decade ago. A number of countries in the tropics, with a tradition of hyperendemic malaria, have now greatly reduced the incidence by modern control measures. The disease is still a major cause of ill-health in many parts of tropical and subtropical Africa, Asia, Haiti, Central and South America, and the Southwest Pacific.
3. **Infectious agents**—*Plasmodium vivax* for vivax malaria, *P. malariae* for quartan malaria, *P. falciparum* for falciparum malaria, and *P. ovale* for the less common ovale malaria.
4. **Reservoir and source of infection**—Man is the only important reservoir, but higher apes may harbor *P. malariae*. The immediate source of infection is an infective mosquito.
5. **Mode of transmission**—Certain species of *Anopheles* ingest human blood containing plasmodia in the gametocyte stage and act as definitive hosts. The parasite develops into sporozoites in from 8 to 35 days depending on species of parasite and temperature to which the insect is exposed. Sporozoites concentrate in the salivary glands and are injected into man as the insect thereafter takes blood meals. In the susceptible host, gametocytes usually appear in the blood within 3 to 14 days, according to species of parasite. Malaria may be transmitted also by injection or transfusion of blood of infected persons or by use of contaminated hypodermic syringes, as by drug addicts.
6. **Incubation period**—Average 12 days for *P. falciparum*, 14 days for *P. vivax* and *P. ovale*, and 30 days for *P. malariae*. With some strains of *P. vivax*, primary attack may be delayed for 8 to 10 months, the period of latency being known as protracted incubation period. With infection by blood transfusion, incubation is usually short.
7. **Period of communicability**—As long as infective gametocytes circulate in the blood; varies with species and strain of parasite and with response to therapy. May extend indefinitely in quartan malaria, from 1 to 3 years in vivax, and rarely more than 1 year in falciparum. The infected mosquito remains infective for the rest of its life of a month or more; only the female takes blood meals.
8. **Susceptibility and resistance**—Susceptibility is universal; the degree of susceptibility is sometimes lessened by previous infection. A high degree of tolerance to the effects of infection may develop in highly endemic primitive communities where exposure to infective anophelines is continuous over many years.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Application of residual insecticide (such as DDT, benzene hexachloride, or dieldrin) in suitable formula and dosage at appropriate intervals on the inside walls of dwellings and on other surfaces upon which vector anophelines habitually rest will generally result in effective malaria control. Entire communities desirably are in-

cluded in a spraying project, to be carried forward year after year until malaria ceases to be endemic. Country-wide effort over 4 consecutive years has in some instances eradicated malaria.

Both malaria control and malaria eradication are based mainly on the use of residual insecticides, though in some situations administration of antimalarial drugs may be necessary to speed up elimination of the parasite reservoir in man. The techniques of malaria control and malaria eradication are similar, but the acceptable standards are different.

2. Where residual insecticide is not available, nightly spraying of living and sleeping quarters with a liquid or an aerosol preparation of pyrethrum is useful.
3. In endemic areas, living and sleeping quarters should be screened properly and bed nets used.
4. Insect repellents (such as diethyltoluamide, 50% solution, or dimethylphthalate; or 2-ethylhexane-diol, 1, 3, commonly called "612") applied to uncovered skin and impregnated in the clothing of persons exposed to bites of vector anophelines are useful.
5. Sanitary improvements, such as filling and draining, to eliminate breeding places of vector anophelines should not be neglected. Larvicides (such as oil and Paris green) are now not commonly used where residual spraying is effective but may have usefulness under special conditions.
6. Regular use of suppressive drugs in highly malarious areas has special value (see 9B8).
7. Effective treatment of acute and chronic cases is an important adjunct to malaria control and usually is essential in attempted eradication.
8. Antimalarial projects profit materially from health education that teaches modern drug treatment and suppression, and practical measures of prevention.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory case report, Class 2B (p. 5), in nonendemic areas, desirably limited to authenticated cases (USA); Class 3C (pp. 5, 6) is the more practical procedure in endemic areas.
2. Isolation: None; patients desirably are protected at night by screens or bed nets in areas where vector anophelines are present.
3. Concurrent disinfection: A single concurrent residual spraying of the neighborhood may be useful if a primary or relapsing case occurs in an area not under control, previously free from the disease and where potential vectors are active.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Determine history of previous infection or of exposure to infection by anophelines or otherwise.
8. Specific treatment: (a) Acute cases in nonimmune subjects: Chloroquine diphosphate or sulfate 600 mg (base) immediately, followed in 6 hours by 300 mg (base), and 300 mg (base) on each of next 2 days; or amodiaquin dihydrochloride dihydrate, same regimen and dosage as above. (b) Acute cases, emergency treatment: Mepa-

crine methane sulphonate 300 mg intramuscularly, repeated after 6 hours if necessary; or chloroquine hydrochloride, 10 ml of a 5% solution (300 to 400 mg base) intramuscularly, repeated after 6 hours if the patient is not able to take additional drug (i.e. diphosphate) by mouth; or quinine hydrobromide or dihydrochloride 600 mg well diluted in a liter of normal saline, glucose saline, or plasma, administered intravenously and slowly, and repeated after 6 hours if necessary; or chloroquine hydrochloride intravenously, 400 mg base in 500 ml saline, once in 24 hours. (c) Acute cases in semi-immune subjects: Chloroquine diphosphate or sulfate in a single dose 600 mg (base), or amodiaquin dihydrochloride dihydrate, in a single dose 600 mg (base).

Suppression: Chloroquine diphosphate or sulfate, 300 mg (base) once or twice weekly or amodiaquin dihydrochloride dihydrate, 400 mg (base) once weekly, preferably on the same day each week. For semi-immune subjects, pyrimethamine, 25 mg once weekly, or proguanil monohydrochloride 300 mg once weekly.

Prevention of relapse: Primaquine diphosphate, 15 mg (base) daily for 14 days; should be given to persons leaving endemic areas at time suppressive drugs are discontinued; in acute attacks, may be given concurrently with standard chloroquine or amodiaquin treatment.

The above doses are for adults. Children require lesser amounts in relation to age and weight.

C. Epidemic measures: A field survey to determine nature and extent of the hyperendemic or epidemic situation is the point of departure. Intensify residual spraying, treatment of acute cases, and use of suppressive drugs. Sometimes the breeding places of anophelines responsible for an epidemic can be eliminated.

D. International measures:

1. Disinsectization of aircraft, ships or other vehicles on arrival in an area freed from malaria or any of its vectors, if the health authority at arrival has reason to suspect importation of malaria vectors.
2. Disinsectization of aircraft before departure from an area where vectors have become resistant to a particular insecticide or insecticides, using an insecticide of a type to which the vectors are still susceptible.
3. Strong effort to maintain rigid antimosquito sanitation within the mosquito flight range of all ports and airports.
4. In special circumstances administration of antimalarial drugs to migrants, seasonal workers or persons taking part in periodic mass movement into an area or country where malaria has been eradicated.

MEASLES

1. **Identification**—An acute highly communicable viral disease with a prodromal stage characterized by catarrhal symptoms and Koplik spots on the buccal mucous membranes. A characteristic dusky-red blotchy rash appears on the third or fourth day affecting face, body, and extremities, sometimes ending in branny desquamation. Leucopenia is usual. Death from uncomplicated measles is rare. Such deaths as occur are usually the result of secondary pneumonia in children aged less than 2 years,

and the aggregate does not exceed 1 per 1,000 cases of measles. Synonyms: Rubeola, Morbilli.

Virus isolation in tissue culture or demonstration of a rise in specific complement-fixing or neutralizing antibodies is possible, but laboratory facilities for these tests are not often available.

2. **Occurrence**—Common in childhood; probably 80 to 90% of persons surviving to age 20 years have had measles; few persons go through life without an attack. Endemic and relatively mild in large metropolitan communities, attaining epidemic proportions about every other year. In smaller communities, and in rural areas, outbreaks tend to be more widely spaced and somewhat more severe. In areas previously free, or in isolated settlements with long intervals between outbreaks as in the Arctic, measles often affects large proportions of the population and fatality is much increased. Prevalent in all seasons except summer, but primarily spring.
3. **Infectious agent**—The virus of measles.
4. **Reservoir and source of infection**—Reservoir is man. Source of infection is secretions of nose and throat of infected persons.
5. **Mode of transmission**—By droplet spread or direct contact with an infected person; indirectly through articles freshly soiled with secretions of nose and throat. One of the most readily transmitted of communicable diseases; in some instances probably air-borne.
6. **Incubation period**—About 10 days from exposure to initial fever, about 14 days until rash appears; uncommonly longer or shorter. Late inoculation with gamma globulin or convalescent serum in attempted passive protection may extend incubation to 21 days.
7. **Period of communicability**—During the period of catarrhal symptoms; usually about 9 days, from 4 days before to 5 days after rash appears.
8. **Susceptibility and resistance**—Practically all persons are susceptible; permanent acquired immunity is usual after attack. Babies born of mothers who have had the disease are ordinarily immune for the first few months of life.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Education as to special danger of exposing young children to those exhibiting any fever or acute catarrhal symptoms, particularly during years and seasons of epidemic measles.
 2. Encouragement by health departments and by private physicians of administration of immune serum globulin (gamma globulin) to infants and children under 3 years of age in families where measles occurs. This product carries no hazard of serum hepatitis.
 3. Experimental measles vaccines are under study.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Obligatory case report in most states and in many countries, Class 2B (p. 5). Early reporting permits better isolation and adequate care for the underprivileged child and provides opportunity for passive protection of contacts.
 2. Isolation: Commonly 7 days from rash to protect the patient against added infection and to limit transfer of measles to susceptible contacts, especially those under 3 years of age.
 3. Concurrent disinfection: All articles soiled with secretions of nose and throat.

4. **Terminal disinfection:** Thorough cleaning.
5. **Quarantine:** Impractical and of no value in large communities. Exclusion of exposed susceptible school children and teachers from school and from all public gatherings until 14 days from last exposure may be justifiable in sparsely settled rural areas. If date of single exposure is reasonably certain, an exposed susceptible child may be allowed to attend school for the first 7 days of incubation. Quarantine of institutions, wards, or dormitories for young children exposed to measles is of value; strict segregation of infants if measles occurs in an institution.
6. **Immunization of contacts:** Administration of gamma globulin to an exposed person within 3 days after first exposure to a known case of measles will avert the attack in most instances and almost certainly modify it. Such passive immunity has a maximum duration of 3 weeks. Given between 4 and 6 days after first exposure, there is reasonable chance of modifying severity of attack and the patient probably will acquire the usual lasting immunity to measles; given after the sixth day, little effect is to be expected. For protection, the dosage of gamma globulin, given shortly after exposure, is 0.1 ml. per pound of body weight; for modification, in amounts of 0.02 ml. per pound of body weight.
7. **Investigation of contacts and source of infection:** Search for exposed susceptible children under 3 years of age is profitable. Carriers are not known to occur.
8. **Specific treatment:** None. Complications should be treated with an appropriate antibiotic or with sulfadiazine.

C. Epidemic measures:

1. Daily examination of exposed children and known susceptible adult contacts, with record of body temperature. Susceptible persons exhibiting a rise of temperature of 0.5°C (1.0°F) or more should be isolated promptly pending diagnosis.
2. Schools should not be closed nor classes discontinued; daily observation of children by physician or nurse should be provided and sick children promptly removed.
3. In institutional outbreaks, administration of gamma globulin to all susceptibles has value in checking spread of infection and in reducing fatality; accept no new admissions and exclude visitors under 16 years of age, whether the measles outbreak is in the institution or in the community. Removal of patients during the preeruptive period may prevent an outbreak.

D. International measures: None.

MENINGITIS, MENINGOCOCCAL

1. **Identification**—An acute bacterial infection characterized by sudden onset with fever, intense headache, nausea and often vomiting, signs of meningeal irritation, and frequently a petechial rash. With modern chemotherapy a fatality less than 5% is usual, supplanting the former 40 to 50%; much variation under both endemic and epidemic conditions. Delirium and coma often appear early; occasional fulminating cases (Waterhouse-Friderichsen syndrome) exhibit signs of collapse and shock from onset. Meningococcemia without extension to the

meninges occurs occasionally and should be suspected in cases of otherwise unexplained acute febrile illness, particularly if associated with skin eruption and high leucocytosis. Synonym: Cerebrospinal Fever.

Meningococci can usually be cultivated from the blood, the spinal fluid, and the nasopharynx. Stained smears from petechiae examined microscopically may reveal the infectious agent.

A group of meningitides originating as secondary manifestations of parameningeal or general infection, commonly related to nose, accessory nasal sinuses, ear, or lung present clinical signs and symptoms ordinarily indistinguishable from those of meningococcal meningitis. The differentiation is bacteriologic. The commoner infectious agents concerned with purulent secondary meningitis are pneumococci, hemolytic and other streptococci, *H. influenzae*, *Staphylococcus aureus*; less commonly, but of increasing frequency in recent years, members of the colon-aerogenes-proteus group, the *Salmonellae*, *Pseudomonas aeruginosa*, and others. A variety of fungus infections occasionally end in meningitis. *H. influenzae* meningitis may also occur as a primary suppurative meningitis, i.e., without evidence of local or general infection other than of the meninges. Epidemiologic behavior and methods of control of the secondary meningitides are to be found in descriptions of the individual diseases, e.g., pneumonia (p. 132), streptococcal infections (p. 177), staphylococcal infections (p. 173), and fungus infections (p. 61). In addition, the variety of infections included in the Aseptic Meningitis Syndrome (p. 39) enter into differential diagnosis, particularly of early meningococcal meningitis, as does meningismus.

2. **Occurrence**—Endemic and epidemic; no limits in geographic distribution; commonly observed in temperate climates, but large epidemics have occurred in hot, dry regions. Sporadic cases occur throughout the year in both urban and rural areas, with greatest incidence during winter and spring. The disease exhibits high incidence at irregular intervals, with epidemic waves usually lasting 2 to 3 years. In adults, more common where living conditions are crowded as in barracks and institutions.
3. **Infectious agent**—*Neisseria meningitidis* (*N. intracellularis*), meningococcus. Group 1 is involved in most epidemics in the United States, Group 2 along with Group 1 in sporadic cases of interepidemic periods.
4. **Reservoir and source of infection**—Reservoir is man: patients and carriers. Source of infection is discharges from nose and throat of infected persons. Carrier prevalence of 25%, or more, may exist without occurrence of cases. During epidemic periods more than half of a military organization may be healthy carriers of the strain of meningococcus responsible for the epidemic.
5. **Mode of transmission**—By direct contact with infected persons and by droplet spread. Indirect transmission through contact with articles freshly soiled with discharges from the respiratory tract of infected persons has little significance because the meningococcus is especially susceptible to chilling and drying.
6. **Incubation period**—Varies from 2 to 10 days, commonly 3 to 4.
7. **Period of communicability**—Until meningococci are no longer present in discharges from nose and mouth of patients. Meningococci usually disappear from the nasopharynx within 24 hours after administration of appropriate chemotherapeutic agent.
8. **Susceptibility and resistance**—Susceptibility to the clinical disease is slight as evidenced by the low ratio of cases to carriers. Younger age groups are more susceptible, but the disease occurs at all ages. Type,

degree, and duration of immunity after attack are unknown; there are no generally accepted methods for inducing artificial immunity.

9. Methods of control—

A. Preventive measures:

1. Education as to personal cleanliness and necessity of avoiding direct contact and droplet infection.
2. Prevention of overcrowding in living quarters, public transportation, working places and especially in barracks, camps, and ships.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory case report in most states and countries, Class 2A (p. 5).
2. Isolation: Until 24 hours after start of chemotherapy.
3. Concurrent disinfection: Of discharges from the nose and throat and of articles soiled therewith.
4. Terminal disinfection: Cleaning.
5. Quarantine: No complete quarantine; surveillance is profitable.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Impracticable.
8. Specific treatment: Sulfadiazine by mouth, or intravenously with proper hydration. Penicillin in large doses or the tetracyclines may be used additionally.

C. Epidemic measures:

1. Increase the separation of individuals and the ventilation of living and sleeping quarters for such groups of people as are especially exposed to infection because of occupation or some necessity of living condition.
2. Chemoprophylaxis serves usefully in control of outbreaks in institutional and military populations where all members of the community may be included and where medical supervision is practicable. Sulfadiazine will lower the carrier rate markedly and limit the spread of the disease (0.5 gm twice daily for children, 1 gm twice daily for adults, in both instances for 2 days).

D. International measures: None.

MONONUCLEOSIS, INFECTIOUS

1. **Identification**—An acute infection with irregular clinical manifestations usually accompanied by characteristic lymphocytosis and heterophile antibodies in the blood. Three clinical varieties have been described, having one or both of the stated characteristics: (1) Glandular fever (Pfeiffer)—enlargement of lymphatic glands and spleen without throat manifestations, common in children; (2) Infectious mononucleosis—continued fever with absent or inconsequential glandular swelling or throat symptoms, common in young adults; (3) Monocytic angina—a characteristic involvement of throat structures with or without lymphatic gland involvement, common in older children and young adults. The three varieties may be manifestations of the same disease, and the clinical features frequently occur as a graded mixture. A rash of variable morphology is an irregular occurrence and jaundice, splenomegaly, and meningococcalitis are sometimes seen. The duration

is from one to several weeks and the disease rarely ends fatally. Synonyms: Glandular Fever, Monocytic Angina.

Laboratory aids to diagnosis include examination of blood smears for abnormal lymphocytes and test for heterophile antibodies. Serologic tests for syphilis may be temporarily positive in this disease.

2. **Occurrence**—Reported from many parts of the world, particularly continental Europe, Great Britain, Australia, and the United States. Observed as isolated cases and in epidemics, and probably much more prevalent and more widely distributed than indicated by reported cases. Epidemics are most frequently recognized in schools and in institutions for children; cases are commonly reported among medical students, nurses, and hospital personnel.
3. **Infectious agent**—Unknown.
4. **Reservoir and source of infection**—Reservoir unknown, presumably man. The likely source of infection is discharges from the respiratory tract.
5. **Mode of transmission**—Unknown; the suggestion is of person-to-person spread by direct contact.
6. **Incubation period**—Unknown; seemingly varies from 4 to 14 or more days.
7. **Period of communicability**—Undetermined.
8. **Susceptibility and resistance**—Susceptibility apparently is general, but incidence greatest among children and young adults. Mild unrecognized cases probably occur. The degree of immunity conferred by an attack is undetermined.
9. **Methods of control**—
 - A. *Preventive measures*: None.
 - B. *Control of patient, contacts, and the immediate environment*:
 1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6).
 2. Isolation: None.
 3. Concurrent disinfection: Of articles soiled with nose and throat discharges.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: For the individual case, of little value.
 8. Specific treatment: None.
 - C. *Epidemic measures*: Field investigation of epidemics should be undertaken with the hope of adding to knowledge of the disease.
 - D. *International measures*: None.

MUMPS

1. **Identification**—An acute viral infection of sudden onset characterized by fever and by swelling and tenderness of one or more salivary glands, usually the parotid, sometimes the sublingual or submaxillary glands. Involvement of ovaries and testicles is more frequent in persons past puberty; involvement of the central nervous system is not infrequent.

early or late in the disease. Orchitis and meningoencephalitis due to mumps virus sometimes occur without involvement of a salivary gland. Death from mumps is exceedingly rare. Synonym: Infectious Parotitis.

Hemagglutination and complement-fixation tests are of value in identifying atypical forms of infection. The virus may be found in the saliva, blood, and cerebrospinal fluid.

2. **Occurrence**—Clinical disease is less frequent than with other common communicable diseases of childhood, such as measles and chickenpox; many inapparent infections. Winter and spring are seasons of greatest prevalence; sporadic and epidemic except in large cities where the disease is endemic. Outbreaks are frequent and serious in aggregations of young adults, especially military.
3. **Infectious agent**—The virus of mumps.
4. **Reservoir and source of infection**—Man is the reservoir; source of infection is saliva of infected persons.
5. **Mode of transmission**—By droplet spread and by direct contact with an infected person, or indirectly through articles freshly soiled with the saliva of such persons.
6. **Incubation period**—From 12 to 26 days, commonly 18 days.
7. **Period of communicability**—From about 7 days before distinctive symptoms and persisting as much as 9 days thereafter, but no longer than swelling of a salivary gland. Susceptibles may contract the disease through exposure to persons with inapparent infection.
8. **Susceptibility and resistance**—Susceptibility believed to be general. Second attacks are uncommon; immunity generally held to be lifelong and develops after inapparent as well as clinical attack. An allergic tuberculin-like response occurs when inactivated virus is injected intradermally in persons previously infected.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Effective vaccines are available but have limited value because induced immunity probably does not exceed 2 years. Hence, vaccination of children in most instances is contraindicated because enduring immunity from infection is preferable, thus avoiding serious consequences of mumps after adolescence.
 2. Vaccination has value in selected groups of susceptibles in military practice or a labor force likely to be exposed to the disease for a limited time.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
 2. Isolation: For 9 days.
 3. Concurrent disinfection: Of eating and drinking utensils; of articles soiled with secretions of nose and throat.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: Not applicable generally (see 9A1).
 7. Investigation of contacts and source of infection: Not profitable.
 8. Specific treatment: None.
 - C. **Epidemic measures:** No procedures in common use are effective in control of epidemics.
 - D. **International measures:** None.

NOCARDIOSIS

1. **Identification**—A chronic fungus infection, frequently initiated in the lungs, with hematogenous spread to produce peritonitis, meningitis, brain abscess, and other pyogenic lesions; highly fatal.
Microscopic examination of stained smears of sputum, pus or spinal fluid reveals partially acid-fast, branching hyphae; confirmation is by culture and pathogenicity for animals.
Mycetoma, a localized, benign but disabling mycosis, usually of a lower extremity, is caused by some species of *Nocardia*. Mycetomas caused by fungi other than *Nocardia* often are referred to as madura-mycosis (Madura foot); they require differentiation.
2. **Occurrence**—Occasional sporadic infections in all parts of the world. Mycetomas are more common in regions where people go barefoot. No evidence of age, sex, or racial differences.
3. **Infectious agents**—*Nocardia asteroides* causes generalized disease and brain abscess; *N. brasiliensis*, *N. madurae*, and other species of *Nocardia* cause mycetomas.
4. **Reservoir and source of infection**—Reservoir is soil; source of infection is soil or dust.
5. **Mode of transmission**—Direct contact with contaminated soil through minor traumatic wounds and abrasions; pulmonary infections presumably occur through inhalation of organisms suspended in dust.
6. **Incubation period**—Unknown; probably weeks.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—Unknown.
9. **Methods of control**—
 - A. **Preventive measures**: None.
 - B. **Control of patient, contacts, and the immediate environment**:
 1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
 2. Isolation: None.
 3. Concurrent disinfection: Of discharges and contaminated dressings.
 4. Terminal disinfection: Thorough cleaning.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Not profitable.
 8. Specific treatment: Sulfonamides and penicillin are effective in systemic infections if given early and for prolonged periods.
 - C. **Epidemic measures**: Not applicable, a sporadic disease.
 - D. **International measures**: None.

ONCHOCERCIASIS

1. **Identification**—A chronic, nonfatal disease caused by a filarial worm which forms fibrous nodules in skin and subcutaneous tissues, particularly of head and shoulders (America) or pelvic girdle and lower extremities (Africa). The female worm discharges microfilariae which migrate

through the skin. If lesions are on the head, the microfilariae often reach the eye, leading to ocular disturbances and sometimes blindness. Indurated, pigmented, and inflamed lesions of the skin often occur around microfilariae.

Laboratory diagnosis is by superficial biopsy of skin with demonstration of microfilariae in fresh preparations by microscopic examination; by excision of nodule and finding adult worms; in ocular manifestations, by observation with ophthalmic microscope of microfilariae in cornea, anterior chamber, or vitreous body.

2. **Occurrence**—Geographical distribution in Western Hemisphere limited to Guatemala (principally western slope of continental divide), southern Mexico (states of Chiapas and Oaxaca), and northeast Venezuela; in Africa along west coast from Sierra Leone, south to French Equatorial Africa and Angola, and eastward through the Belgian Congo to Nyasaland, Kenya, Uganda, Ethiopia, and the Sudan. In some localities, nearly all of the population is infected.
3. **Infectious agent**—*Onchocerca volvulus*, a filarial nematode worm.
4. **Reservoir and source of infection**—Reservoir is infected persons with microfilariae in the skin; immediate source of infection is the blackfly, *Simulium*.
5. **Mode of transmission**—By the bite of infected blackflies of the genus *Simulium*; in Guatemala and Mexico *S. ochraceum*, *S. callidum*, *S. metallicum*, and possibly other species; in Africa *S. damnosum* and *S. neavei*. The microfilariae penetrate the thoracic muscles of the vector, develop into infective larvae, migrate to the proboscis, and are liberated in the skin of man as the insect feeds; in Africa vector is infective after 6 days or more, in Guatemala measurably longer because of lower temperatures.
6. **Incubation period**—Nodules may be visible 3 to 4 months after infection. Worms begin to discharge larvae 1 year or more after infection.
7. **Period of communicability**—As long as living microfilariae persist in the skin; probably years.
8. **Susceptibility and resistance**—Susceptibility is universal.
9. **Methods of control**—

A. Preventive measures:

1. Avoid bites of *Simulium* flies by covering body and head as much as possible, or by use of insect repellents.
2. Control of vector larvae in rapidly running streams and in artificial waterways by DDT or other insecticide and sometimes of adult *Simulium*; feasibility depends on vector and terrain.
3. Provision of facilities for diagnosis and treatment.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3C (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: A community problem (see 9A, 9C).

8. Specific treatment: Diethylcarbamazine (hetrazan®) is useful, but causes severe reactions due to destruction of larvae. It does not kill adult worms. Suramin sodium (naphuride sodium, Bayer 205®) leads to gradual disappearance of microfilariae and kills worms. Undesirable reactions may occur and require close medical supervision. Neither is suited to mass treatment procedure. Excision of nodules in order to eliminate adult worms.

C. Epidemic measures: In areas of high prevalence make concerted effort to reduce incidence, using measures listed under 9A.

D. International measures: Coordinated programs entered into by neighboring countries where the disease is endemic, designed to prevent migration of infected persons across international boundaries and to institute treatment and other control measures near such boundaries.

PARAGONIMIASIS

1. **Identification**—A trematode infection with clinical manifestations in man dependent upon path of migration and organs parasitized. The lungs are the most frequent site, where worms are surrounded by an inflammatory reaction which eventually organizes into a fibrous cystic lesion. Clinical symptoms are cough and hemoptysis; in many cases roentgenographic findings closely simulate pulmonary tuberculosis. Ectopic development is not infrequent, worms maturing in such sites as the intestine, lymph nodes, genitourinary tract, subcutaneous tissue and brain, the latter often associated with a Jacksonian-type epilepsy. Infections are usually chronic, lasting for many years, and patients may appear surprisingly well. Synonyms: Pulmonary Distomiasis, Endemic Hemoptysis, Oriental Lung Fluke Disease.

The sputum generally contains flecks of orange-brown pigment, sometimes diffusely distributed, in which masses of worm eggs are seen microscopically. Eggs are also found in feces, rarely in urine.

2. **Occurrence**—Extensive in the Far East, particularly Korea, Japan, Taiwan, the Philippines, and parts of mainland China. Scattered foci in Africa, India, and parts of South America.

3. **Infectious agent**—*Paragonimus westermani*, a trematode.

4. **Reservoir and source of infection**—Man, dog, cat, pig, and wild carnivores are definitive hosts and act as reservoirs. Crabs and crayfish, second intermediate hosts, are the immediate source of infection.

5. **Mode of transmission**—Eggs leave the definitive host via sputum and feces, gain entrance to fresh water, and embryonate in 2 to 4 weeks. A larva (miracidium) hatches, and on penetrating a suitable fresh water snail (*Semistulospira*, Orient; *Pomatiopsis*, North America), undergoes a cycle of development which requires approximately 3 months. Larvae (cercariae) emerge from the snail and penetrate and encyst in fresh water crabs (*Eriocheir* and *Potamon*, Orient) and crayfish (*Astacus*, Orient; *Cambarus*, North America). When the flesh of crustacea containing infective larvae (metacercariae) is ingested by a susceptible mammal raw or only partially cooked, the larva emerges in the duodenum, penetrates the intestinal wall, migrates through the diaphragm, and enters the lung where it develops into an egg producing adult. Worms occasionally develop to maturity in various other organs.

6. **Incubation period**—Flukes mature and begin to lay eggs approximately 6 weeks after man ingests infective larvae. The interval until symptoms appear is long, variable, and poorly defined.
 7. **Period of communicability**—Eggs may be discharged by the human host for as long as 20 years or more. Little available information on duration of infection in mollusc and crustacean hosts. Under natural conditions not directly transmissible from man to man.
 8. **Susceptibility and resistance**—Susceptibility is general. Increased resistance possibly develops as a result of infection.
 9. **Methods of control**—
 - A. **Preventive measures:**
 1. Education of people in endemic areas concerning the life cycle of the parasite; stress thorough cooking of crustacea.
 2. Sanitary disposal of sputum and feces.
 3. Control of snails by proper molluscicides is feasible in some areas, as well as destruction of crabs.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3B (pp. 5, 6).
 2. Isolation: None.
 3. Concurrent disinfection: Of sputum and feces.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: None.
 8. Specific treatment: Emetine hydrochloride and chloroquine both produce clinical improvement and an occasional cure.
 - C. **Epidemic measures:** In an endemic area, occurrence of small clusters of cases or even sporadic infections is important indication for examination of local waters for infected snails, crabs and crayfish, and determination of reservoir mammalian hosts.
 - D. **International measures:** None.
-

PARATYPHOID FEVER

1. **Identification**—A generalized bacterial infection, often of abrupt onset, with continued fever, involvement of lymphoid mesenteric tissues and of intestines, enlargement of spleen, sometimes rose spots on trunk, and usually diarrhea. Many mild attacks result in no more than a transient diarrhea. Fatality is much lower than for typhoid fever.

Laboratory confirmation is by bacteriologic examination of blood, feces, and urine.
2. **Occurrence**—Incidence in United States has fallen with that of typhoid fever. Occurs sporadically or in limited outbreaks. Probably more common than reports suggest because of the large number of unrecognized infections. Paratyphoid A infection is rare in United States, but common in Europe; paratyphoid B is common in the United States, often as a transient diarrhea of undetermined etiology; both are still frequent in countries where sanitation is defective. Paratyphoid C infection is extremely rare in United States but common in Eastern Europe and Asia.

3. **Infectious agents**—*Salmonella paratyphi*, *S. schottmuelleri*, *S. hirschfeldii*, (paratyphoid bacilli A, B, and C). Any strain of *Salmonella* may occasionally produce a bacteremic infection with similar clinical reaction. (See Salmonellosis, p. 162).
4. **Reservoir and source of infection**—Reservoir is man; both patients and carriers, with temporary carriers often frequent in epidemics. Source of infection is feces and urine of infected persons.
5. **Mode of transmission**—Direct or indirect contact with patient or carrier. Vehicles of indirect spread are food, especially milk, milk products and shellfish, usually contaminated by hands of a carrier or missed case. Under some conditions, flies are vectors. Few outbreaks are related to water supplies. Most major outbreaks in England in recent years have been associated with synthetic cream.
6. **Incubation period**—One to 10 days; somewhat longer for paratyphoid A than for B and C.
7. **Period of communicability**—As long as the infectious agent continues in excreta; usually from appearance of prodromal symptoms, throughout illness, and for varying periods after recovery.
8. **Susceptibility and resistance**—Susceptibility is general. Some species-specific immunity usually follows recovery. The degree of protection conferred by paratyphoid vaccine is uncertain.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. The preventive measures applicable to paratyphoid fever are those listed under Typhoid Fever 9A, p. 209.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Obligatory case report in most states and countries, both suspect and confirmed infections, Class 2A (p. 6).
 2. Items 9B, 2-8, of Typhoid Fever also apply to paratyphoid fever. Therapeutic results from chloramphenicol are not striking.
 - C. **Epidemic measures:**
 1. The procedures are those of Typhoid Fever 9C, p. 211.
 - D. **International measures:** Inoculation with triple typhoid vaccine (TAB, typhoid, paratyphoid A, paratyphoid B) is advised for international travelers unless protected through previous attack; not a legal requirement of any country. The risk is minimal in the United States, Canada, Great Britain and Northwest Europe.

PEDICULOSIS

1. **Identification**—Infestation of the scalp, of the hairy parts of the body, or of clothing, especially along the seams of inner surfaces, with adult lice, larvae, or nits. Synonym: Lousiness.
2. **Occurrence**—World-wide. The head louse is common in outbreaks among school children.

3. **Infesting agents**—*Pediculus humanus*, head louse or body louse, and *Phthirus pubis*, crab louse. Animal lice do not infest man.
4. **Reservoir and source of infestation**—Reservoir is infested persons. Sources of infestation are such persons themselves and their personal belongings, particularly body clothing or infested bedding.
5. **Mode of transmission**—Direct contact with an infested person and indirectly by contact with clothing and headgear of such persons.
6. **Incubation period**—Under optimum conditions the eggs of lice hatch in a week, and sexual maturity is reached in approximately 2 weeks.
7. **Period of communicability**—While lice remain alive on the infested person or in his clothing, and until eggs (nits) in hair and clothing have been destroyed.
8. **Susceptibility and resistance**—Any person may become lousy under suitable conditions of exposure. Repeated infestations often result in dermal hypersensitivity.

9. **Methods of control**—

A. Preventive measures:

1. Direct inspection of the heads and, when necessary, of the body and clothing particularly of children in schools, institutions, and summer camps.
2. Education in the value of using hot water and soap to maintain cleanliness, and laundering of clothing to destroy nits and lice.
3. Provision of residual insecticide for freeing persons and clothing of lice. (See 9B8.)

B. Control of infested person, contacts, and the immediate environment:

1. Report to local health authority: Official report not ordinarily justifiable; school authorities should be informed, Class 5 (p. 6).
2. Isolation: Not necessary after application of effective insecticide.
3. Concurrent disinfection: Of other members of family or associated group.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Does not apply.
7. Investigation of contacts and source of infestation: Examination of household and other close personal contacts, with concurrent treatment as indicated.
8. Specific treatment: 10% DDT dusting powder for body and head lice; dust clothing, particularly along seams, and the hair; cover head with towel or cap for several hours; comb hair with fine tooth comb; repeat dusting in 1 week without washing hair or clothing in the interim. For crab lice, dust hairy parts of body and bathe after 12 to 24 hours; repeat treatment in 1 week; continue treatment at weekly intervals until lice or nits are no longer present. Gamma isomer of benzene hexachloride (lindane) as a 1% dusting powder or a 1% ointment, Kwell,® may be substituted; is lethal to both lice and their eggs. Shampoo followed by application of 1% gamma benzene hexachloride in cream or lotion is effective and acceptable for head lice.

C. Epidemic measures: Mass treatment as recommended in 9B8.

D. International measures: None.

PHLEBOTOMUS FEVER

1. **Identification**—A 3- or 4-day fever clinically not unlike influenza except for absence of inflammation of the respiratory tract. Headache, fever of 38.3°C to 39.5°C (101°F to 104°F), retrobulbar pain on motion of the eyes, injected sclerae, malaise, and pain in the limbs and back are characteristic. Leucopenia is usual, most prominent on fourth or fifth day after onset of fever. Temperature occasionally exceeds 104°F and the disease may present alarming symptoms, but death is unknown. Diagnosis is clinical and by epidemiological means, through occurrence of multiple and similar cases. One of the group of Arthropod Borne Viral Fevers, p. 33. Synonyms: Pappataci Fever, Sandfly Fever, Three-day Fever.

Diagnosis may be confirmed by neutralization test, using mouse adapted viruses, or by isolation of virus in newborn mice.

2. **Occurrence**—In those parts of Europe, Africa, and Asia where the vector exists; not recognized with certainty in Central or South America. A disease of subtropical and tropical areas having long periods of hot, dry weather; in general, a belt extending around the Mediterranean and eastward into Burma and China. Seasonal, between April and October, and prone to appear as a disease of troops and travelers from nonendemic areas.

3. **Infectious agents**—The viruses of phlebotomus fever; at least 2 immunological types have been identified.

4. **Reservoir and source of infection**—Man is one reservoir; an animal reservoir is suspected but not demonstrated. The immediate source of infection is an infective *Phlebotomus*.

5. **Mode of transmission**—The vector is a small, hairy, blood-sucking midge, *Phlebotomus papatasi*, the common sandfly, which bites at night and has a limited flight range; other species of *Phlebotomus* may carry the virus.

6. **Incubation period**—Up to 6 days, usually 3 to 4 days, rarely less.

7. **Period of communicability**—Virus is present in the blood of an infected person at least 24 hours before and 24 hours after onset of fever. *Phlebotomus* becomes infective about 7 days after biting an infected person and remains so for life.

8. **Susceptibility and resistance**—Susceptibility is essentially universal; homologous acquired immunity is lasting. There is no heterologous immunity. Relative resistance of native populations in sandfly areas is probably attributable to infection early in life.

9. **Methods of control**—

A. Preventive measures:

1. Control of sandflies is the important consideration. For detailed procedures, see Bartonellosis, p. 41.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3C (pp. 5, 6).

2. Isolation: None; protect infected individual from bites of *Phlebotomus* for first few days of illness, by screening or mosquito bed nets (25–30 mesh to the inch) or by spraying quarters with insecticide.

3. Concurrent disinfection: None; destruction of sandflies in the dwelling.

4. Terminal disinfection: None.

5. Quarantine: None.

6. Immunization of contacts: None currently available.
7. Investigation of contacts and source of infection: Search for breeding areas of sandflies around dwellings, especially in rubble heaps, masonry cracks, and under stones.

8. Specific treatment: None.

C. Epidemic measures:

1. Community use of insecticide to destroy the *Phlebotomus* midge in and about human habitations.
2. Public health education on the conditions under which infection is acquired; the necessity to avoid bites of *Phlebotomus* by use of repellents while in infected areas, particularly after sundown; and to enlist cooperation with public health authorities in efforts to eliminate *Phlebotomus*.

D. International measures: None.

PINTA

1. **Identification**—An acute and chronic nonvenereal treponematosiis, characterized clinically by a superficial nonulcerative primary lesion, a secondary eruption of flat erythematous lesions known as pintids, and late dyschromic changes in the skin. Within 7 to 20 days after inoculation a scaling papule appears, usually on hands, legs or dorsum of feet, and having a satellite bubo. In 5 to 12 months a maculopapular, erythematous secondary rash appears and may evolve into tertiary lesions, the dyschromic stage, with achromic or pigmented (blue, pink, yellow, violet) spots of variable size, mainly on distal portions of extremities but often including trunk and face. In rare instances the untreated disease may end fatally. Synonyms: Mal del Pinto, Carate, Tina, Lota, Empeines, Azul, and others.

Serologic tests for syphilis usually become reactive during the secondary rash and thereafter behave as in venereal syphilis.

2. **Occurrence**—Frequent among dark-skinned people of tropics and subtropics; in Western Hemisphere among Negroes, native Indians, and those of mixed blood. Especially prevalent in Mexico, Colombia, Venezuela, and Ecuador. Pinta-like conditions are reported from east and west coasts of Africa, North Africa, the Middle East, and in India and the Philippines. Predominantly a disease of childhood.
3. **Infectious agent**—*Treponema carateum*.
4. **Reservoir and source of infection**—Reservoir is man; source of infection is principally initial skin lesions and those of early dyschromic stage of infected persons.
5. **Mode of transmission**—Unknown; evidence suggests transmission by direct and indirect contact; location of primary lesions suggests an influence by trauma. Various biting and sucking arthropods have been implicated; rare reports of venereal and congenital transmission.
6. **Incubation period**—Seven to 20 days.
7. **Period of communicability**—Unknown; potentially communicable while skin lesions are active, sometimes for many years.
8. **Susceptibility and resistance**—Undefined; presumably as in other treponematoses. Rare in white persons, suggesting some inherent insus-

ceptibility, but not distinguished clearly from factors of personal hygiene and social and economic status.

9. Methods of control—

- A. *Preventive measures:* Those applicable to other nonvenereal treponematoses apply to pinta. (See Yaws 9A, p. 222.)
- B. *Control of patient, contacts, and the immediate environment:*
 - 1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3B (pp. 5, 6).
 - 2. Items 2 to 8, see Yaws 9B (pp. 222, 223).
- C. *Epidemic measures:* See Yaws 9C.
- D. *International measures:* See Yaws 9D.

PLAGUE

1. **Identification**—A severe and highly fatal disease with severe toxemia, high fever, shock, fall in blood pressure, rapid and irregular pulse, restlessness, staggering gait, mental confusion, prostration, delirium, and coma. Conjunctival injection is common; hemorrhages may occur. Intense pain in groin, armpit, or neck with appearance of bubo. Sporadic cases are mainly of 2 clinical forms: (a) Bubonic plague is the more common, with acutely inflamed and painful swellings of lymph nodes draining the site of original infection. Secondary invasion of the blood stream often leads to localization of infection in diverse parts of the body. A secondary, often terminal pneumonia has special significance as the means by which primary pneumonic plague is engendered. (b) Septicemic plague, proved by blood smear or blood culture, is rare; it is a form of bubonic plague in which the bubo is obscure. During outbreaks two other forms are recognized: (c) Primary pneumonic plague and (d) Tonsillar septicemic plague. Though uncommon, primary pneumonic plague occurs in localized and sometimes devastating epidemics among groups of persons closely associated. Untreated bubonic plague has a fatality of 25 to 50%; primary septicemic plague and pneumonic plague are usually fatal. Modern therapy materially reduces the fatality of bubonic plague; pneumonic plague also responds if recognized and treated early.

Clinical diagnosis is confirmed by demonstrating the infectious agent in fluid aspirated from buboes, in blood, or in sputum of severe or pneumonic plague.

2. **Occurrence**—Sylvatic (wild rodent) plague exists in the western third of the United States, in large areas of South America, in Central and in Southern Africa, in the Near East with a center in Iranian Kurdistan, also the frontier area between Yemen and Saudi Arabia, and in Central Asia. The foci in Southeast Russia are apparently quiescent. Plague in man in the United States is limited to rare instances of exposure to wild rodents or their fleas. Urban plague has been largely controlled in most of the world; even rural bubonic plague of rat origin, until recently a serious health problem in India, Burma, and Indonesia (Java), presently shows a marked decline. Plague nevertheless continues potentially dangerous because of vast areas of persisting sylvatic infection.
3. **Infectious agent**—*Pasteurella pestis*, plague bacillus.
4. **Reservoir and source of infection**—Wild rodents are the natural reservoir of plague; numerous species in many parts of the world are

subject to periodic epizootics (sylvatic or wild rodent plague). The infection may transfer to domestic rats in urban or rural areas where rats and wild rodents have common contact (urban plague). The immediate source of infection in bubonic plague of man is an infective flea; in pneumonic infection, exhaled droplets and sputum of patients.

5. **Mode of transmission**—Bubonic plague is transmitted by the bite of an infective (blocked) rat flea, *Xenopsylla cheopis* and certain other species. Pneumonic plague and tonsillar plague are spread by person-to-person contact from patients with primary pneumonic plague or from patients with bubonic plague who develop terminal plague pneumonia. Accidental infections occur among laboratory workers.
6. **Incubation period**—From 2 to 6 days in bubonic plague; 3 to 4 days in pneumonic plague; may be shorter, rarely longer.
7. **Period of communicability**—Bubonic plague under natural conditions is not directly transmissible from person to person except through terminal plague pneumonia. Fleas may remain infected for days or weeks under suitable conditions of temperature and humidity, or may clear themselves of infection; infective (blocked) fleas are usually extremely short-lived (3 to 4 days). Pneumonic plague is usually intensely communicable under climatic or social conditions which lead to overcrowding in unsanitary dwellings.
8. **Susceptibility and resistance**—Susceptibility is general. Bubonic infection is occasionally no more than a localized infection of short duration, pestis minor. Immunity after recovery is temporary and relative. Active immunization with a vaccine of killed bacteria may confer considerable protection for some months when administered in 2 or 3 doses at weekly intervals; repeated stimulating injections are necessary for continued protection. Vaccines prepared with living avirulent strains may confer satisfactory immunity in one dose, repeated once yearly.

9. Methods of control—

A. Preventive measures:

1. Periodic surveys in endemic and potentially epidemic areas to determine the prevalence of rats and rat fleas; suppression of rats by poisoning or trapping in urban areas. Continuing inspection and survey of wild rodents and their ectoparasites in areas of sylvatic plague. Where plague is present or threatening, systematic search for evidence of infection in rodents and their fleas by pooling methods.
2. Ratproofing of buildings and reduction of breeding places and harborage, particularly on docks and in warehouses.
3. Rat control on ships by ratproofing or periodic fumigation, combined when necessary with destruction of rats and their fleas in vessels and cargoes arriving from plague localities.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report of suspect and confirmed cases universally required by international regulation. Class 1 (p. 5).
2. Isolation: Hospitalize all patients if practical; ordinary aseptic precautions suffice for patients with bubonic plague; strict isolation is required for primary pneumonic plague or patients developing plague pneumonia.
3. Concurrent disinfection: Sputum and purulent discharges, and articles soiled therewith; urine and feces of patients.

4. Terminal disinfection: Thorough cleaning; bodies of persons dying of plague should be handled with strict aseptic precautions.
5. Quarantine: For contacts of bubonic plague, disinfestation with insecticide powder such as 5 to 10% DDT in talc or pyrophyllite and surveillance for 6 days; for contacts of pneumonic plague, dust with insecticide powder, quarantine for 6 days with close observation for developing illness, including recording of body temperature every 4 hours; start specific therapy as soon as fever appears.
6. Immunization of contacts: None; the management of contacts should concentrate on surveillance, quarantine, and close observation with prompt institution of specific treatment at first appearance of symptoms of fever. When circumstances or resources do not permit close observation of contacts, chemoprophylaxis may be necessary (see 9B-8).
7. Investigation of contacts and source of infection: Search for infected rodents and fleas or exposure to preceding cases of plague pneumonia or pneumonic plague.

Focal attack on fleas should precede anti-rat measures, using an appropriate insecticide powder with residual effect such as 10% DDT in talc or pyrophyllite. Dust rat runs and harborages in and about known or suspected plague premises or areas. Disinfect by dusting or insecticide spray the houses, outhouses and household furnishings in the same areas. Dust the persons and clothing of immediate contacts and all other residents in the immediate vicinity. Supplemental suppression of rat populations by poisoning or trapping then follows.

8. Specific treatment: Streptomycin, the tetracyclines, and chloramphenicol when used early are highly effective in all forms of plague. Results are good even in pneumonic plague if therapy is begun within 24 hours of onset, but poor thereafter. Recurrence of fever during streptomycin therapy may indicate that the causative organisms are resistant to the drug or that a complication has developed, such as secondary pneumonia due to other bacteria. In this event penicillin or other appropriate antibiotic is indicated. Penicillin is not effective against plague itself. Recommended dosages for treatment are as follows: Streptomycin, 1.0 gm intramuscularly followed by 0.5 gm every 4 hours until temperature remains normal for 4 or 5 days. Broad spectrum antibiotics, 2.0 to 4.0 gm daily beginning with a loading dose of at least 1.0 gm by the parenteral route. When antibiotics are not available, sulfonamides should be used, sulfadiazine or its equivalent, 1.0 gm every 4 hours.

For chemoprophylaxis when necessary, daily doses for broad spectrum antibiotics should be at least 1.0 gm per day, and for sulfonamides 2.0 to 3.0 gm per day, for 6 days.

C. Epidemic measures:

1. Investigate all deaths, with autopsy and laboratory examinations when indicated. Develop case-finding facilities. Establish the best possible provision for diagnosis and treatment. Alert all existing medical facilities toward immediate reporting and toward utilization of diagnostic and therapy services. Provide adequate laboratory services and supplies of antibiotics.
2. Institute intensive flea control in expanding circles from known focal areas.
3. Supplemental rat destruction within affected areas.

4. Prophylactic administration of broad spectrum antibiotics or sulfadiazine to all medical, nursing, and public health personnel exposed to definite and repeated risk of infection may be considered if they cannot be kept under close and frequent observation to detect disease in its earliest stages. Quarantine of hospital personnel to the premises is desirable when patients with pneumonic plague are under treatment.
5. Personal protection of field workers against fleas by weekly dusting of clothing with insecticide powder. Daily application of insect repellents is a valuable adjunct.
6. Widespread active immunization of native populations with a single dose of avirulent living plague vaccine has proved valuable. Killed vaccines are less useful in such circumstances because of practical difficulties in administering repeated injections.

D. International measures:

1. Telegraphic notification of WHO and adjacent countries by governments of a first case of plague in man, the origin of which is within the country, or of newly discovered or reactivated foci of plague among rodents.
2. Measures applicable to ships, aircraft, and land transport arriving from plague areas are specified in International Sanitary Regulations, World Health Organization, annotated edition, Geneva, 1957.
3. All ships should be periodically deratted, or kept permanently in such condition that rat populations are reduced to a minimum.
4. Ratproofing of buildings of ports and airports; application of appropriate insecticide with residual effect every 6 months; deratting with effective rodenticide.
5. International travelers: Plague infected persons or suspects are not permitted to depart a country. No country currently requires immunization against plague for entry. Because of the short duration of protection from vaccines, the recommendation in the face of an existing epidemic or anticipated unusual exposure is for immunization on arrival.

PLEURODYNIA

1. **Identification**—An acute infectious disease characterized by sudden onset of severe paroxysmal pain commonly localized at the costo-diaphragmatic border, and accompanied by intermittent fever, headache, anorexia, and malaise. Duration 1 to 3 days; remissions frequent. A nonfatal illness usually recognized in localized epidemics. Synonyms: Epidemic Pleurodynia, Bornholm Disease, Epidemic Myalgia, Devil's Grippe.
Diagnosis may be aided by isolation of Cocksackie Group B viruses from feces and by demonstrating a rise in titer of type-specific neutralizing antibodies in blood serums of early and late illness. Differential count of blood leucocytes is usually normal.
2. **Occurrence**—The disease is not common; usually occurs in epidemics, with outbreaks reported in Europe, England, Australia, New Zealand, and North America. A summer and early autumn disease, occurring at all ages but most commonly manifest in children and young adults. Multiple cases in a household are frequent.
3. **Infectious agents**—Various Cocksackie Group B viruses. Types 1, 3, and others have been associated repeatedly with the illness.

4. **Reservoir and source of infection**—Reservoir is man. Source of infection is feces and throat discharges of persons ill with pleurodynia. Coxsackie Group B viruses Types 1 and 3 also are present in well persons in an epidemic area, and occasionally in persons with illnesses not typical of pleurodynia.
5. **Mode of transmission**—Probably contact with an infected person or with articles freshly soiled with infective material. Coxsackie Group B viruses have been found in sewage, on flies, and mosquitoes; the relation to transmission of human disease is not clear.
6. **Incubation period**—Three to 5 days is usual.
7. **Period of communicability**—Unknown, but apparently during the acute stage of disease.
8. **Susceptibility and resistance**—Susceptibility is probably general, and presumably a type-specific immunity results from infection.
9. **Methods of control**—
 - A. **Preventive measures:** None.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6).
 2. Isolation: None.
 3. Concurrent disinfection: Prompt and safe disposal of nose and throat discharges and of feces. Articles soiled therewith should be disinfected.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Of no practical value.
 8. Specific treatment: None.
 - C. **Epidemic measures:**
 1. General notice to physicians of the epidemic occurrence of the disease and the necessity for differential diagnosis of illness which might mistakenly be considered a medical or surgical emergency.
 - D. **International measures:** None.

PNEUMONIA

A. PNEUMOCOCCAL—ACUTE LOBAR PNEUMONIA

1. **Identification**—An acute bacterial infection characterized by sudden onset with chill followed by fever, often pain in chest, and usually a productive cough, dyspnea, and leucocytosis. Roentgenographic examination may disclose pulmonary lesions prior to other evidence of consolidation. Not infrequently pneumonia is bronchial rather than lobar, especially in children, with vomiting and convulsions the first manifestations. Pneumococcal pneumonia is an important cause of death, generally and among acute infections. Fatality is greatly reduced by antibiotic and chemotherapy; formerly 20 to 40% for hospital patients, but now ordinarily a small fraction of those rates; much variation according to serologic type of pneumococcus and age of patient, being highest for infants and the aged.

Laboratory confirmation is by bacteriological examination of sputum or of discharges of the respiratory tract. A rise in antibody titer between acute-phase and convalescent-phase serums is useful in problem cases, and culture of the blood in severe infections.

2. **Occurrence**—A common disease affecting a large proportion of the population, particularly in infancy and old age. No race, color, or sex is exempt. Most frequent in industrial cities and lower economic groups. Occurs in all climates and seasons, most often in winter and spring in temperate zones, and in regions where cold, windy, changeable and inclement weather prevails. Usually sporadic in the United States, but epidemics occur in institutions and in barracks; consistently recurring epidemics have been described in South African mines. A rising incidence is commonly associated with influenza epidemics.
3. **Infectious agent**—*Diplococcus pneumoniae*. Pneumococci Types I to XXXII account for about 95% of cases; the remainder are due to rarely recognized types.
4. **Reservoir and source of infection**—Reservoir is man; patients and carriers. Pneumococci may be found in the upper respiratory tract of healthy members of most communities throughout the world. Source of infection is respiratory secretions.
5. **Mode of transmission**—By droplet spread, by direct contact with patients or carriers, or indirectly through articles freshly soiled with discharges of nose and throat of such persons. Transmission by air-borne particles may be possible but has not been established as important. Person-to-person transmission of the pneumococcus is common, but secondary cases among contacts and attendants are infrequent.
6. **Incubation period**—Not well determined; believed to be 1 to 3 days.
7. **Period of communicability**—Unknown; presumably until the discharges of mouth and nose no longer carry the infectious agent in appreciable numbers or in virulent form. Penicillin will eliminate the pneumococcus from most patients within 3 days.
8. **Susceptibility and resistance**—Resistance is generally high but may be lowered by wet, cold and exposure, and apparently under certain conditions by physical and mental fatigue, and by alcoholism. Inapparent infection is common, particularly with Type 3 pneumococci and strains of higher types. Immunity to the homologous type of pneumococcus usually follows an attack, may last for months or years, and is highly specific. Active immunization against specific types is possible but rarely practical.
9. **Methods of control**—
 - A. **Preventive measures**:
 1. Whenever practicable and particularly in institutions, barracks, and on shipboard, crowding in living and sleeping places should be avoided. General resistance should be conserved by good food, fresh air, sufficient sleep, temperance in the use of alcoholic beverages, and other hygienic measures.
 2. Chemoprophylaxis with sulfonamide or antibiotic drugs is feasible for closed populations in time of epidemics, but has not been adequately evaluated.
 3. Active immunization with bacterial vaccines or polysaccharides of prevailing types of pneumococci may be effective for the control of epidemics in limited populations such as mine workers and military units.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6). Reported deaths are a better index of frequency of the disease.
2. Isolation: None.
3. Concurrent disinfection: Of discharges from nose and throat.
4. Terminal disinfection: Thorough cleaning and airing.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Of no practical value.
8. Specific treatment: Penicillin intramuscularly; oral penicillin G is effective. The tetracycline antibiotics produce comparable results. In event of penicillin sensitivity or delayed response to penicillin, sulfonamide drugs and erythromycin are usually effective.

C. Epidemic measures:

1. In outbreaks in institutions or in other limited or closed population groups, general hygienic measures may be supported by chemoprophylaxis with sulfonamides or antibiotics.

D. International measures: None.**B. BACTERIAL PNEUMONIA, OTHER THAN PNEUMOCOCCAL**

1. **Identification**—An acute febrile disease with pulmonary involvement evidenced by symptoms, physical signs, or roentgen-ray examination. Often occurs in association with other infections of the respiratory tract, particularly epidemic influenza. Fatality with adequate treatment is low but appreciable, and variable according to infectious agent and age of the patient.

Appropriate bacteriologic examination of sputum, nasopharyngeal swabs, and blood aid materially in diagnosis.

2. **Occurrence**—World-wide in distribution and a frequent disease in infancy and old age, and in winter months in temperate climates. No racial selectivity. Usually sporadic, but epidemics occur in association with influenza, measles, and other respiratory infections.
3. **Infectious agents**—Various pathogenic bacteria of the mouth, nose, and throat, as *Streptococcus pyogenes* (Group A hemolytic streptococci) *Staphylococcus aureus*, *Klebsiella pneumoniae* (Friedlander bacillus) and *Hemophilus influenzae*.
4. **Reservoir and source of infection**—Reservoir is man; patients and carriers. Source of infection is discharges from mouth and nose.
5. **Mode of transmission**—By droplet spread, by direct contact with patient or carrier, or indirectly through articles freshly soiled with discharges of nose or throat of such persons.
6. **Incubation period**—Variable, usually short, 1 to 3 days.
7. **Period of communicability**—Unknown; probably while the infectious agent is present in discharges of nose and throat of patients. For many of the agents involved, antibiotic therapy greatly decreases the period of communicability.
8. **Susceptibility and resistance**—Susceptibility appears to be low grade; highest in infants and young children, and in the aged. Immunity varies with the infectious agent and is probably minimal except for

type-specific immunity to Group A streptococci. Immunization procedures are not feasible.

9. Methods of control—

A. Preventive measures:

1. Good personal hygiene; avoid crowding in institutions and hospitals.
2. Immunization against influenza (p. 94) and chemoprophylaxis of streptococcal infections (p. 177) may be applied to limited or general populations.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6). Identification of a preceding respiratory infection is of public health significance.
2. Isolation: None.
3. Concurrent disinfection: Of discharges from mouth and nose and of articles soiled therewith.
4. Terminal disinfection: Thorough cleaning and airing.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Of no practical value.
8. Specific treatment: Streptococcal: Same as pneumococcal pneumonia. Staphylococcal: Procaine penicillin; if organism proves resistant, tetracycline antibiotics as in pneumococcal pneumonia; sensitivity tests with the organism isolated may aid in selecting the most suitable antibiotic. *H. influenzae*: The tetracyclines and chloramphenicol are effective; combined sulfadiazine-streptomycin is also used. *K. pneumoniae* (Friedlander bacillus): Streptomycin in the acute phase, then tetracyclines or chloramphenicol.

C. Epidemic measures:

1. Applicable only in outbreaks in institutions or in other limited or closed population groups when associated with influenza, measles, or other respiratory infection. Active immunization against influenza and passive immunization of infants and children against measles may be employed. Chemoprophylaxis may be effective but has not been adequately evaluated.

D. International measures: None.

C. PRIMARY ATYPICAL PNEUMONIA (VIRUS PNEUMONIA)

1. **Identification**—An acute respiratory infection characterized by gradual and insidious onset, constitutional symptoms of chilliness, feverishness, headache, malaise and fatigue, and respiratory symptoms of cough and expectoration. Initial physical signs in lungs are minimal but develop later. Early patchy infiltration, demonstrable by roentgenographic examination of chest, is often more extensive than clinical findings suggest. Numbers and distribution of leucocytes are usually normal. Duration of illness averages about a week and complications are infrequent; fatality is about 0.1%.

Development of cold hemagglutinins during convalescence or of agglutinins for *Streptococcus MG*, or both, confirms diagnosis in one-half to two-thirds of cases. Such cases may constitute an etiological entity differing from those cases not showing these agglutinins.

Differentiation required from psittacosis, influenza, Q fever, adenovirous infections and a number of other infections with similar clinical manifestations.

2. **Occurrence**—World-wide distribution, as a sporadic, endemic, and occasionally epidemic disease especially in institutions and military populations. Reliable attack rates for civilian populations are not known: rates of 10 per 1,000 per annum are recorded among military populations. Incidence greatest during winter months in temperate zones, with much variation from year to year and in different geographic areas. No selectivity for race or sex. Occurs at all ages, but recognized disease is more frequent among adolescents and young adults.
3. **Infectious agent**—Most cases apparently due to a virus or viruses not yet isolated or characterized, or given a specific name.
4. **Reservoir and source of infection**—Reservoir is man; patients and persons with mild, unrecognized infections. Source of infection is discharges from mouth and nose.
5. **Mode of transmission**—By intimate contact with a patient or with articles freshly soiled with discharges of nose and throat of such person.
6. **Incubation period**—Believed to be 7 to 21 days, commonly 12.
7. **Period of communicability**—Unknown; presumably during late incubation and throughout febrile illness.
8. **Susceptibility and resistance**—The low incidence in general populations and low attack rate among contacts suggest a relatively high resistance. Most infected persons suffer no more than mild respiratory illness with no pneumonia. Degree and duration of immunity after attack are unknown; second attacks are rare. No available immunization.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. When possible, crowding in living and sleeping quarters should be avoided, especially in institutions, in barracks, and on shipboard. General resistance should be maintained by adequate food, sufficient sleep, fresh air, and good personal hygiene.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6).
 2. Isolation: None.
 3. Concurrent disinfection: Of discharges from nose and throat.
 4. Terminal disinfection: Thorough cleaning and airing.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Of no practical value.
 8. Specific treatment: Tetracycline antibiotics have been reported to give beneficial results in individual cases. Symptomatic and supportive treatment is effective in most instances.
 - C. **Epidemic measures:** No certainly effective measures for control are available. Isolation precautions in epidemic situations may be helpful.
 - D. **International measures:** None.

POLIOMYELITIS

1. **Identification**—An acute illness characterized by fever, malaise, headache, stiffness of neck and back, and moderately increased cells and protein of the spinal fluid. Paralysis of voluntary muscles, most commonly of lower extremity, occurs in severe cases; many infections of the non-paralytic type are mild and some are indistinguishable from the Aseptic Meningitis Syndrome, p. 39; others have vague symptoms and no signs referable to the central nervous system. Inapparent infection exceeds clinical cases at least a hundredfold. Fatality varies from 2 to 10% and for bulbar poliomyelitis from 5 to 60%. Synonym: Infantile Paralysis.

Paralytic poliomyelitis ordinarily is identifiable clinically, but non-paralytic poliomyelitis usually requires laboratory tests for exact recognition. The virus of poliomyelitis can be isolated by tissue culture from feces or throat secretions during early and current infections; a rising titer of complement-fixing or neutralizing antibodies denotes recent infection.

Other nonsuppurative, mainly viral, infections of the central nervous system (Arthropod-borne Encephalitides, p. 30; Encephalitis, other forms, p. 30, Sect. 1 Par. 3; Lymphocytic Choriomeningitis, p. 108; Aseptic Meningitis Syndrome, p. 39; also Syphilitic Meningitis, p. 185, and Tuberculous Meningitis, p. 203) are to be differentiated from non-paralytic poliomyelitis. Together these conditions account for a considerable proportion of cases reported as nonparalytic poliomyelitis, especially in nonepidemic times.

2. **Occurrence**—Poliomyelitis infection occurs throughout the world. The clinical disease occurs sporadically and in epidemics, with incidence highest in summer and early autumn but varying widely from year to year and region to region. Large areas may experience low incidence for several years with an ultimate reappearance of high incidence. Paralytic disease is more frequent in temperate zones. Children from 1 to 16 years of age are more frequently attacked than adults, but in several areas including the United States, the proportion of cases among older children and young adults is greater than formerly. In densely populated areas, especially tropical or subtropical, where crowding prevails and sanitation is poor, the age pattern of poliomyelitis differs and points to usual infection with all 3 types of virus early in life; clinical paralytic disease is there confined mainly to the first 5 years, is relatively infrequent, epidemics are few, and serological studies reveal the general presence of antibodies after the first few years of life. Age of initial infection of population groups is in a general way related to social status. In countries where artificial immunization has been widely practiced, occurrence of paralytic cases and of epidemics has been concentrated among the least vaccinated population groups.
3. **Infectious agent**—Poliovirus, Types 1, 2, and 3; readily isolated by tissue culture and distinguishable immunologically.
4. **Reservoir and source of infection**—Reservoir is man, most frequently persons suffering from clinically unrecognized or inapparent infections, especially children. Source of infection is pharyngeal secretions and feces of infected persons.
5. **Mode of transmission**—Direct contact and droplet spread through close association with infected persons. In rare instances milk has been a vehicle. No reliable evidence of spread by other foods, insects, or sewage; water is rarely involved. Whether the feces or respiratory secretions of an infected person have the greater importance in transmis-

sion has not been determined. Virus is more readily detectable and for a longer period in feces than in the throat, but there is epidemiologic evidence to suggest that respiratory spread is important, especially where good sanitation is practiced.

6. **Incubation period**—From 3 to 21 days, commonly 7 to 12 days.
7. **Period of communicability**—Communicability is at a maximum level in late incubation and early days of acute illness, virus being present in throat secretions and feces; persists in feces for 3 to 6 weeks or longer but spread of infection after the acute stage is rare.
8. **Susceptibility and resistance**—Susceptibility to infection is general but few infected persons develop paralysis. Type-specific resistance of long duration follows both clinically recognizable and inapparent infection. Second attacks are rare and presumably due to infection with a different type. Infants born of immune mothers have transient passive immunity. Removal of tonsils, recent or remote, predisposes to bulbar involvement. Injections of precipitated antigens and certain other substances may precipitate paralysis in an already infected but otherwise symptomless person, the paralysis being characteristically confined to or appearing first in the injected limb. Excessive muscular fatigue in the prodromal period may likewise predispose to paralytic involvement. An increased susceptibility to paralytic poliomyelitis appears to be associated with pregnancy.

9. Methods of control—

A. Preventive measures:

1. Active immunization with formalinized virus (Salk vaccine) reduces the risk of paralytic disease, but does not prevent infection or excretion of virus by those infected. The recognized series of inoculations is 3 injections, the first 2 about 4 weeks apart, the third about 7 months after the second. Duration of protection is still to be determined. A fourth injection one year or more after the third is recommended. Immunization of all persons in susceptible age groups is the desired objective, but in community programs priority should be given to ages with highest incidence and to selected groups at unusual risk. Immunization of infants may begin at age 2 months. General community programs are best instituted during seasons of low incidence, so timed that the final injection just precedes seasonal prevalence. Vaccines of living attenuated virus are being evaluated in the laboratory and in the field.
2. Immune serum globulin contains antibodies against poliomyelitis virus but is of little practical value in passive protection of communities. (See 9B6 below.)

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory case report in most states and countries, Class 2A (p. 5). Each individual case is to be designated as paralytic or nonparalytic poliomyelitis, avoiding type unspecified in order to facilitate essential epidemiologic analyses.
2. Isolation: For 1 week from date of onset, or for duration of fever if longer. Of little value because spread of infection is greatest in the prodromal period and because of the many unrecognized infections present in a community.
3. Concurrent disinfection: Of throat discharges and feces and of articles soiled therewith. In communities with modern and adequate sewage disposal systems, feces and urine can be disposed of directly into sewer without preliminary disinfection.

4. Terminal disinfection: Cleaning.
5. Quarantine: Of unproved value. Restriction of family contacts is theoretically useful but of no practical benefit because of large numbers of unrecognized infections in the community.
6. Immunization of contacts: Vaccination of familial and other close contacts contributes little to immediate control; ordinarily the virus is widely spread among such contacts by the time the first case is recognized. Passive protection with gamma globulin may be expected to prevent only an occasional case in an exposed family; in instances of known single exposure at a susceptible age, may have considerable value, if given within 2 days after exposure.
7. Investigation of contacts and source of infection: Thorough search for sick persons, especially children, to assure treatment of unrecognized and unreported cases.
8. Specific treatment: None; attention to prevention and management of paralysis.

C. Epidemic measures:

1. Mass vaccination should be undertaken at the earliest indication of an epidemic, but cannot be expected to have an immediate effect; it is no substitute for routine immunization. The value of live vaccines in outbreak control is being explored. Mass passive immunization is not practical.
2. Protection of children so far as practicable against unnecessary close contact with persons outside the family. Urban schools should not be closed or opening delayed, but intensive or competitive athletic programs should be postponed. In time of epidemic rural schools, especially where buses are used to gather children from sparsely populated areas, and boarding schools which draw children from areas free of the disease and at a distance, should not be opened until the epidemic subsides.
3. Postponement of elective nose and throat operations.
4. Postponement of inoculation of any precipitated type antigen unless risk of disease for which immunization is intended is considered greater than the slightly increased risk of poliomyelitis.
5. Avoidance of excessive physical strain as in violent exercise during epidemic or after known exposure.
6. Avoidance of unnecessary travel and visiting, especially of children, during high prevalence of infection.
7. Isolation in bed of all children with fever, pending diagnosis.
8. Education in such technics of bedside nursing and clinical care as will restrict development of deformities and reduce risk of transmission of infection.
9. Provision for prolonged care of paralyzed cases to provide maximum recovery through rehabilitation therapy.

D. International measures:

1. Telegraphic notification of epidemics by national health authorities to WHO.
2. WHO designated poliomyelitis centers provide countries with epidemiological information needed to design sound programs of control, ascertain the prevalence of virus types and the state of immunity of populations. They act as reference laboratories for the identification of poliomyelitis and related viruses.

PSITTACOSIS

- 1. Identification**—An acute generalized infectious disease of viral origin. Onset is with fever and headache, early pneumonic involvement, cough initially absent or nonproductive, later usually present and productive; sputum mucopurulent, never copious; anorexia extreme; commonly constipation; pulse usually slow in relation to temperature; lethargy; occasional relapses. Normal or slightly increased numbers of leucocytes early, leucopenia later. Treatment with broad spectrum antibiotics has produced a drastic decline in fatality; less than 1% for reported cases in the United States. Deaths are mainly among persons more than 30 years old, and among infants. Human infections are frequently mild, particularly those of pigeon and duck origin. Synonym: Ornithosis.
Laboratory diagnosis is by demonstrating significant increase in complement-fixing antibodies during convalescence. Isolation of virus from sputum, blood or post-mortem tissues is possible.
- 2. Occurrence**—World-wide distribution, as sporadic cases or household outbreaks among persons exposed to sick or seemingly healthy birds. In the United States an occupational disease of pet shops, aviaries, pigeon lofts, poultry farms, and poultry processing plants.
- 3. Infectious agent**—A virus of the psittacosis-ornithosis group, antigenically related to the virus of lymphogranuloma venereum. Other related viruses have been identified in mammalian hosts: cattle, sheep, goats, and cats; their relationship to human disease is not determined.
- 4. Reservoir and source of infection**—Reservoirs are parakeets, parrots, pigeons, turkeys, finches, petrels, domestic fowl, and other birds; occasionally man. Birds apparently healthy can be carriers and occasionally shed virus. Sources of infection are cloacal excretions, feathers and viscera of birds and sputum of infected persons.
- 5. Mode of transmission**—Contact with infected birds, chiefly household pets, or their recent surroundings; also pigeon lofts, squab farms, turkey and duck raising farms and processing plants. Transmission from man to man is rare. Virus is probably air-borne. Laboratory infections are frequent. Infection by ingestion is unproved.
- 6. Incubation period**—From 4 to 15 days, commonly 10 days.
- 7. Period of communicability**—Primarily during the acute illness, especially with paroxysmal coughing. Diseased birds may shed virus throughout infection and sometimes for weeks or months if not properly treated.
- 8. Susceptibility and resistance**—Susceptibility is general, but older adults have a more severe disease; one attack does not always confer immunity.
- 9. Methods of control**—
 - A. Preventive measures:**
 1. Strict regulation of import or traffic in birds of parrot family based on quarantine and laboratory examination.
 2. Quarantine of pet shops where psittacosis has occurred. Infected birds should be destroyed and the premises thoroughly cleaned. In some instances birds can be treated, but they should remain in quarantine until evidence that they are healthy.
 3. To protect human contacts, psittacine birds offered in commerce should be raised under disease-free conditions and handled in such manner as to prevent infection.
 4. Education of the public in the danger of household or occupational exposure to infected birds of the parrot family.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory case report in most states and countries, Class 2A (p. 5).
2. Isolation: Important during acute febrile stage. Nurses caring for patients with a cough should wear adequate masks.
3. Concurrent disinfection: Of all discharges.
4. Terminal disinfection: Thorough wet cleaning and exposure to sunlight. Buildings housing infected birds should not be used by human beings until thoroughly cleaned and disinfected; formalin is effective.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Trace source of suspected birds. Infected birds should be killed and bodies immersed in 2% cresol. Spleen, liver, and kidneys should be removed aseptically while feathers are wet, frozen in sterile container, and sent to nearest available laboratory. Carcasses should be burned after autopsy.
8. Specific treatment: Tetracycline antibiotics, continued for at least one week after temperature returns to normal.

C. Epidemic measures: Ordinarily not applicable to man because cases are usually sporadic or confined to small family outbreaks. The epidemic problem is with birds. Report outbreaks of ornithosis in flocks of turkeys to state health authority, which has the obligation to determine virulence of the infectious agent, and by serologic tests ascertain the frequency of infection. Large doses of tetracyclines will suppress but not eliminate infection. Flocks infected with virus of low virulence for mice may be processed under supervision if so directed by state health and animal disease departments. Destroy flock when virulence is high, visceral lesions are present in 40 to 50% of birds and antibiotic treatment is uneconomical. Identify susceptibles among employees on farms and in processing plants by serologic tests; by choice, workers should be restricted to those with demonstrated antibodies.

D. International measures: Reciprocal respect for national regulations designed to control importation of psittacine birds.

Q FEVER

1. Identification—Q fever is characterized by sudden onset, chilly sensations, retrobulbar headache, weakness, malaise, severe sweats, and much variation in severity and duration. Pneumonia, similar to that of atypical pneumonia, occurs in most cases along with mild cough, scanty expectoration, chest pain, minimal physical findings, and little or no upper respiratory involvement. Chronic general infections have been reported. The fatality before introduction of specific therapy was not more than 1%; it is now negligible except in aged persons.

Laboratory diagnosis is by complement-fixation or agglutination tests, with demonstration of rise in antibody titer between acute and convalescent stages; or by recovery of the infectious agent from blood of the patient, readily accomplished but hazardous to laboratory workers.

2. **Occurrence**—Reported from all continents. More widespread in the United States than generally recognized. Endemic in California where infection exists enzootically in animals raised for meat and milk; has occurred in explosive epidemics in a number of areas in United States, among workers in diagnostic laboratories, stock yards, meat packing and rendering plants, and wool processing factories.
3. **Infectious agent**—*Rickettsia burneti* (Coxiella burneti).
4. **Reservoir and source of infection**—Ticks, wild animals (bandicoots), cattle, sheep, and goats are natural reservoirs, with infection usually inapparent. The common source of infection is dust contaminated by products of domestic animals, particularly placental tissues, fetal membranes, amniotic fluid and milk. Other sources of infection are bodies of patients, animal carcasses, contaminated wool, straw, and laundry. Ticks are rarely the source of human infection.
5. **Mode of transmission**—Commonly by air-borne dissemination of rickettsiae, in or near contaminated premises, in establishments processing infected animals and at autopsy. Raw milk from infected cows may be responsible for some cases; also contracted by direct contact with infected animals or with other contaminated materials.
6. **Incubation period**—Usually 2 to 3 weeks.
7. **Period of communicability**—Under natural conditions direct transmission from man to man is negligible.
8. **Susceptibility and resistance**—Susceptibility is general. An attack confers immunity for at least a year. A suitable vaccine induces immunity.
9. **Methods of control**—

A. Preventive measures:

1. Immunization with inactivated vaccine prepared from *R. burneti* infected yolk sac is so useful in protecting laboratory workers that it should be considered for others in hazardous occupations.
2. Pasteurization of milk from cows, goats and sheep at 62.9° C (145° F) for 30 minutes, or at 71.7° C (161° F) for 15 seconds by the high-temperature short-time method, or boiling of milk, inactivates rickettsiae.
3. Public education on sources of infection and the necessary hygienic practices of pasteurization of milk and adequate disposal of animal placentas.
4. Control infection of animals by vaccination and by regulating movement of infected livestock. The control of Q Fever in man depends upon control of the disease in animals.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas (USA): in many states and countries not a reportable disease, Class 3B (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: Of sputum, excreta and blood, and articles freshly soiled therewith.
4. Terminal disinfection: None; precautions at post-mortem examination.
5. Quarantine: None.
6. Immunization of contacts: Unnecessary.
7. Investigation of contacts and source of infection: Search for history of contact with cattle, sheep, and goats, consumption of raw milk, or direct or indirect association with a laboratory handling *R. burneti*.

8. Specific treatment: The tetracycline antibiotics or chloramphenicol, administered orally and continued for several days after afebrile; reinstitute if relapse occurs.
 - C. **Epidemic measures:** Outbreaks are generally of short duration; control measures are essentially limited to observation of exposed persons and antibiotic therapy for those becoming ill. In hyperendemic situations immunization should be considered for persons at greatest risk.
 - D. **International measures:** Control of importation of domestic animals, especially goats, sheep, and cattle.
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RABIES

1. **Identification**—An almost invariably fatal acute encephalitis. Onset is with a sense of apprehension, headache, fever, malaise, and indefinite sensory changes often referred to the point where virus entered. The disease progresses to paresis or paralysis, with spasm of muscles of deglutition on attempt to drink. Delirium and convulsions follow; death is from respiratory paralysis. Usual duration is from 2 to 6 days, sometimes longer. Synonym: Hydrophobia.
2. **Occurrence**—Uncommon in man; primarily a disease of animals. Occurs throughout the world except in Australia, New Zealand, Hawaii, and other Pacific Islands, some of the West Indies, Great Britain, and the Scandinavian peninsula; absent for many years in Denmark, Netherlands, Belgium, and Switzerland. Urban rabies is a problem of dogs and occasionally other pets; sylvatic or rural rabies primarily involves wild biting animals, with sporadic infection of dogs and domestic livestock.
3. **Infectious agent**—The virus of rabies.
4. **Reservoir and source of infection**—Reservoirs include a large group of wild and domestic *Canidae*, including dog, fox, coyote, wolf, and also cat, skunk, raccoon, and other biting mammals. Vampire and fruit eating bats are infected in South and Central America and Mexico; infection of insectivorous bats recently recognized in the United States, Canada, Europe, and the Middle East. The source of infection in rabies of man is the saliva of rabid animals.
5. **Mode of transmission**—By bite of a rabid animal or rarely by saliva of such animals entering a scratch or other fresh break in the skin. Transmission from man to man conceivably is possible but has not been confirmed; the saliva is known to be infectious.
6. **Incubation period**—Usually 2 to 6 weeks, occasionally longer; depends on extent of laceration, site of wound in relation to richness of nerve supply and other factors.
7. **Period of communicability**—In the dog and most biting animals, for 3 to 5 days before onset of frank clinical signs, and during the course of the disease. Bats may shed virus for many months.
8. **Susceptibility and resistance**—Mammals are generally susceptible. Natural immunity is unknown in man. Antirabic vaccination induces active immunity in man and dogs.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Detention and clinical observation for 10 days of dogs and other animals known to have bitten a person, or having suspicious signs

of rabies. Such animals should not be killed until active rabies is established, preferably by a veterinarian. Rabid animals usually have a change in behavior, with excitability or paralysis, and followed by death; if animal is infective at the time of bite, signs and symptoms of rabies will follow within 10 days except for bats.

2. Immediate destruction or 6 months detention in approved pound or kennel of unvaccinated dogs or cats bitten by known rabid animals. If animal has previously been vaccinated, revaccinate and restrain for 30 days.
3. Immediate submission to a laboratory of iced intact heads of animals dying of suspected rabies for laboratory examination; confirmation of rabies is by demonstration of Negri bodies in nerve cells of the brain and by animal inoculation with demonstration of virus.
4. Wounds of persons caused by bite or scratch of an animal with rabies or suspected rabies should be cleaned promptly with soap or detergent solution. Hyperimmune serum may be infiltrated beneath the bite wound. The wound should not be sutured for at least several days.
5. Specific prevention of rabies in man is by vaccination of persons bitten by a known or suspected rabid animal. Protection depends on administration of the vaccine soon after injury, usually on 14 consecutive days, in severe or multiple bites sometimes for 21 days. Vaccination often is supplemented by passive immunization with hyperimmune serum. The following is a guide to procedure in different circumstances:
 - a. If the animal is apprehended, confine and observe it for 10 days. Give vaccine to exposed person at first physical sign or laboratory evidence of rabies in the observed animal.
 - b. If animal is not apprehended and rabies is known to be present in the area, vaccination should be started immediately.
 - c. In severe bites, particularly in region of head, face and neck, where there is any likelihood of the animal being rabid, give a dose of rabies hyperimmune serum immediately; follow promptly with a full course of vaccine. An intradermal sensitivity test should precede administration of serum.
 - d. Rabies vaccination carries a small risk of postvaccinal encephalitis. This must be weighed against the hazard of contracting rabies. Vaccine should not be given unless the skin is broken. The schedule of inoculations may be reduced if the person has had a previous series of antirabic inoculations. If severe sensitivity appears in the course of vaccination, either with suspensions of nervous tissue or avianized (duck egg) vaccine, complete the series with the other type.
6. A system of registration and licensing of dogs favors control measures. All owned dogs in congested areas should be kept on leash when not within homes of owners. Stray dogs should be collected and destroyed by public authority. Emphasis on preventive vaccination of dogs; attenuated live vaccines administered intramuscularly confer longer lasting immunity than inactivated vaccines.
7. Cooperative programs with wildlife conservation authorities toward reducing numbers of foxes, skunks, and other wildlife hosts in areas of sylvatic rabies.
8. Education of the public in the necessity of complying with restrictions on dogs, of vaccinating dogs, of seeking immediate medical atten-

tion if bitten by a dog, of confining and observing animals that have inflicted bites, of prompt reporting to the police of dogs manifesting strange behavior, and of reporting rabies in dogs and bites of persons by animals to the local health authority.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory case report required in most states and countries, Class 2A (p. 5).
2. Isolation: For the duration of the illness.
3. Concurrent disinfection: Of saliva and articles soiled therewith. Immediate attendants should be warned of the hazard of inoculation with saliva and provided with rubber gloves and protective gowns.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Contacts of a patient with rabies need not be vaccinated.
7. Investigation of contacts and source of infection: Search for rabid animal and for persons and other animals bitten.
8. Specific treatment: For clinical rabies, none.

C. Epidemic measures:

1. Establishment of area control under authority of state laws, public health regulations and local ordinances, in cooperation with appropriate wildlife conservation and livestock sanitary authorities.
2. Widespread vaccination of dogs, preferably with an attenuated live vaccine, through officially sponsored intensified programs providing mass immunization at temporary and emergency stations.
3. Strict enforcement of regulations requiring collection, detention and destruction of ownerless or stray dogs, and of unvaccinated dogs found off owner's premises.

D. International measures: Strict compliance by common carriers and by travelers with national laws and regulations that institute quarantine or require vaccination of dogs.

RAT-BITE FEVER

Two diseases are included under the general term of rat-bite fever; one, also known as Haverhill Fever, is caused by *Streptobacillus moniliformis*; the other, also known as Sodoku, is caused by *Spirillum minus*. Because of similarity in clinical and epidemiological behavior, and because *Streptobacillus moniliformis* infection is more common in the United States, it alone is presented in detail. Individual variations manifested by *Spirillum minus* infection are noted under that disease.

A. STREPTOBACILLUS MONILIFORMIS INFECTION

1. **Identification**—Usually a history of rat bite within 10 days; primary edematous lesion; swelling of regional lymph nodes; sharp febrile paroxysms often alternating with afebrile intervals, and accompanied by a morbilliform and petechial rash, polyarthritides, and leucocytosis. The bite wound commonly heals by first intention, later to break down leaving an ulcer that runs a relapsing course, often painful and prolonged; the result sometimes is a subcutaneous abscess. Fatality may reach 10% in untreated cases. Synonym: Haverhill Fever.

Confirmation is by bacteriologic examination of primary lesion, lymph nodes, blood, joint fluids and pus, by specific agglutination test, or by mouse inoculation. Caution should be exercised lest the experimental mouse or rat be already naturally infected.

2. **Occurrence**—Distribution is world-wide; an uncommon disease in North and South America and in most European countries. Information based on case reports shows this to be the usual form of rat-bite fever in the United States.
3. **Infectious agent**—*Streptobacillus moniliformis* (*Streptothrix muris rattis*, *Haverhillia multiformis*, *Actinomyces muris*).
4. **Reservoir and source of infection**—Reservoir is an infected rat, rarely other rodents (squirrel, weasel). Source of infection is secretions of mouth, nose, or conjunctival sac of infected animals. Sporadic cases without reference to bite have been recorded.
5. **Mode of transmission**—By the bite of an infected rodent; animal blood escapes from the injured or diseased buccal mucosa into the wound, or contamination is by conjunctival secretions. Blood from an experimental laboratory animal has infected man. Actual contact with rats is not necessary; infection has occurred in persons working or living in rat-infested buildings. A few localized epidemics have been traced to contaminated milk or milk products (Haverhill fever); the means of contamination is not known, whether through infection of cows by rat bite, or direct contamination of milk by rats.
6. **Incubation period**—Three to 10 days, rarely longer.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—No information concerning man.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Reduction of rat population. Ratproofing of dwellings.
 2. Pasteurization of milk may help in preventing Haverhill fever.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6).
 2. Isolation: None.
 3. Concurrent disinfection: None.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Not practicable.
 8. Specific treatment: Tetracyclines or penicillin. Penicillin inhibits only the bacillary form and has no effect on the L_1 phase of the *Streptobacillus*. Treatment should continue for 7 to 10 days.
 - C. **Epidemic measures:** Grouped cases presenting the typical symptoms require search for epidemiologic evidence of a relation to milk supply.
 - D. **International measures:** None.

B. SPIRILLUM MINUS INFECTION

A sporadic rat-bite fever, Sodoku, is caused by *Spirillum minus* (*Spirocheta morsus muris*). It is less frequently observed in the United States than *Streptobacillus moniliformis* infection, but in Japan and the Far East is the common form. Incidence is no greater there than in western countries. The fatality is

approximately 10%. Clinical *Spirillum minus* infection differs from *Streptobacillus* infection in the usual absence of arthritic symptoms, and a more plaque-like rash. The incubation period is generally longer, 1 to 3 weeks, and usually more than 7 days. Laboratory methods are essential for differentiation of the 2 diseases.

RELAPSING FEVER, LOUSE-BORNE

1. Identification—An epidemic spirochetal disease with short febrile paroxysms lasting 2 to 3 days, alternating with afebrile periods of 3 to 4 days and resulting in 2 to 10 or more relapses, more commonly the lesser number. Each attack terminates by crisis and the average duration of the disease is 13 to 16 days. Transitory petechial rashes are common during the initial fever. Fatality is between 2 and 10%.

Diagnosis is by demonstration of the infectious agent in darkfield preparations of fresh blood, stained thick blood films, or by intraperitoneal inoculation of white rats with 15 to 25 ml of blood taken during the pyrexial period and before crisis.

2. Occurrence—In limited localities in Europe and Asia, in East Africa (Kenya and Ethiopia), North and South Africa, and in Central America. Epidemics are commonly incident to war and famine or other situations where malnourished, overcrowded populations with poor personal hygiene favor multiplication and wide dissemination of the vector. Not reported in the United States for many years.

3. Infectious agent—*Borrelia recurrentis*, a spirochete. Numerous specific names have been given to morphologically identical but biologically different spirochetes isolated from cases of louse-borne relapsing fever in widely separated endemic areas.

4. Reservoir and source of infection—Reservoir is man, with the spirochete present in the blood of infected persons; interepidemic reservoir is unknown. Immediate source of infection is an infective louse.

5. Mode of transmission—By crushing an infective louse, *Pediculus humanus*, into the bite-wound or into an abrasion of the skin.

6. Incubation period—Up to 12 days, usually 7 days.

7. Period of communicability—Under natural conditions not directly transmissible from man to man. The louse becomes infective 4 to 5 days after ingestion of blood from an infected person and remains so for life (20 to 40 days). Man is infective for louse mainly during pyrexia.

8. Susceptibility and resistance—Susceptibility is general. The duration of immunity after clinical attack is unknown but probably does not exceed 2 years.

9. Methods of control—

A. Preventive measures:

1. Routine application of insecticide with residual effect, such as DDT or a number of other compounds, at appropriate intervals to populations living under conditions favoring the development of lousiness.
2. Individual prophylaxis through application of insecticide, DDT or equivalent, at appropriate intervals as by dusting powder or by impregnation of clothing.
3. Improvement of living conditions with provision for frequent bathing and washing of clothing.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report universally required by International Sanitary Regulations, Class 1 (p. 5).
2. Isolation: None, provided the patient, his clothing, all household contacts and the immediate environment have been deloused (9A2).
3. Concurrent disinfection: None, if proper delousing has been done.
4. Terminal disinfection: Careful terminal application of insecticides to body and clothing of patient where death occurs before this has been done.
5. Quarantine: Exposed louse-infested susceptibles may be released after application of insecticide with residual effect; otherwise quarantine for 15 days.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: For the individual case, unprofitable; a community effort (see 9C).
8. Specific treatment: Penicillin G in adequate dosage; tetracyclines and chloramphenicol are also effective. Patients with central nervous system involvement, particularly if dosage of drug is inadequate, will likely experience relapse. Arsenical therapy, neoarsphenamine and mapharsen®, is widely used.

C. Epidemic measures:

1. The most important measure for the rapid control of relapsing fever, where reporting has been good and the number of cases small, is application of insecticides with residual effect to contacts of all reported cases. Where infection is known to be widespread, systematic application of residual insecticide to all persons in the community is indicated (2 to 5% DDT in an inert powder, talc or pyrophyllite for dusting).

D. International measures:

1. Telegraphic notification of WHO and of adjacent countries, by governments, of the existence of an epidemic of relapsing fever. An internationally quarantinable disease.
2. Measures applicable to ships, aircraft, and land transport arriving from relapsing fever areas are specified in International Sanitary Regulations, World Health Organization, annotated edition, Geneva, 1957.
3. International travelers: Patients or suspects are not permitted to leave an area until judged free of infection, or of infestation with lice. Health authorities in endemic or epidemic regions may require disinfection before departure of other travelers considered liable to spread relapsing fever; the same requirement holds on arrival at destination.

RELAPSING FEVER, TICK-BORNE

1. **Identification**—Regularly an endemic disease with a clinical course similar to that of the louse-borne disease, except that relapses are more frequent; deaths are rare. A satisfactory history of tick bite is unlikely and evidence of bite is seldom found.

Diagnosis is by demonstrating the infectious agent in thick smears

of blood at time of febrile attack, or by inoculation of mice, rats, or monkeys with blood drawn at that time.

2. **Occurrence**—Widespread throughout tropical Africa. Foci have been observed in Spain, North Africa, Saudi Arabia, Iran, India, and parts of Central Asia as well as in North and South America. In the United States human cases of tick-borne relapsing fever have been found in limited localities of many western states.
3. **Infectious agent**—*Borrelia duttoni*, a spirochete. Numerous specific names have been given to morphologically identical but biologically different spirochetes isolated from cases of tick-borne relapsing fever in widely separated endemic areas.
4. **Reservoir and source of infection**—Wild rodents are the natural reservoir. Immediate source of infection is an infective argasid tick.
5. **Mode of transmission**—Man is infected by the bite or coxal fluid of an infected tick, principally 1 of 5 species of the genus *Ornithodoros*; in the United States, *O. turicata* and *O. hermsi*; *O. rudis* and *O. talaje* are vectors in Central and South America; *O. moubata* in tropical Africa; and *O. tholozani* in Near East, Middle East, and Far East. The tick attacks, rapidly engorges and promptly leaves the body of the host. Transovarian infection occurs in ticks.
6. **Incubation period**—From 3 to 6 days, but may be as short as 2 days or as long as 12.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Infectivity of man for tick is mainly during febrile attack. Infected ticks can live without feeding for years and remain infective.
8. **Susceptibility and resistance**—Susceptibility is general. The duration of immunity after recovery is indefinite, but probably not more than 2 years.
9. **Methods of control**—
 - A. **Preventive measures**:
 1. Avoid tick-infested caves, camp sites, shacks, and ground areas. If exposure is unavoidable, use tick repellent on exposed areas of skin, (dimethylphthalate, indalone, Rutgers 612, or a number of other compounds now under investigation). Clothing may be impregnated with repellent by spraying or by dipping.
 - B. **Control of patient, contacts, and the immediate environment**:
 1. Report to local health authority: In selected endemic areas (USA); in many countries not a reportable disease, Class 3B (pp. 5, 6).
 2. Isolation: None.
 3. Concurrent disinfection: None.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Important.
 8. Specific treatment: See treatment of Louse-borne Relapsing Fever, 9B8 (p. 148).
 - C. **Epidemic measures**: Reduction of tick population in living quarters by periodic dusting with insecticides. Use repellents.
 - D. **International measures**: None.

RHEUMATIC FEVER

1. **Identification**—Rheumatic fever is an occasional sequel of Group A hemolytic streptococcal upper respiratory infection; sometimes occurs in the absence of such recognized prior infection. The main clinical manifestations are migratory polyarthritides, carditis, chorea, subcutaneous nodules, and erythema marginatum. Fever, rapid pulse, nontraumatic epistaxis, abdominal and precordial pain, pallor, anorexia, weight loss, a fast sedimentation rate, leucocytosis, and electrocardiographic changes are a second group of findings of lesser diagnostic significance. With a history of previous attack, combinations of the above suggest recurrence of rheumatic fever. Mild and inapparent infections occur, their relative frequency unknown; definite and even severe heart disease develops in the absence of evident acute rheumatic fever. An important cause of death among children of the United States, aged 6 to 10 years; the fatality is appreciable, commonly 3 to 5% in endemic areas. **Synonym:** Acute Articular Rheumatism.

Bacteriologic or serologic (chiefly antistreptolysin O) evidence of a preceding Group A streptococcal infection adds diagnostic weight to suggestive symptoms.
2. **Occurrence**—A frequent disease in temperate zones throughout the world; in the United States most prevalent in Rocky Mountain region, New England, and North and Central Atlantic States; lowest in the South and Southwest. Seasonal incidence is that of streptococcal infections, in the United States predominantly during spring months and low during summer and early autumn. Reliable data on frequency in tropical areas are not available; the impression of a lesser prevalence than in temperate zones is not always supported. Predilection for race or sex has not been defined. For unknown reasons, incidence and mortality of rheumatic fever are declining.
3. **Infectious agent**—Unknown. Attacks are usually precipitated by Group A streptococcal respiratory infections, frequently unrecognized or so mild as to have had no medical attention.
4. **Reservoir and source of infection**—Unknown.
5. **Mode of transmission**—Unknown.
6. **Incubation period**—Not applicable. Symptoms appear about 2 to 3 weeks after a recognized Group A streptococcal infection.
7. **Period of communicability**—Not known to be communicable; the preceding streptococcal infection which may precipitate rheumatic fever is communicable but usually has subsided by the time rheumatic fever develops.
8. **Susceptibility and resistance**—All ages are susceptible; the greatest incidence is in children 6 to 12 years old. The disease has a natural tendency to recur with each succeeding Group A streptococcal infection; no evidence that immunity develops although various antibodies to Group A streptococci or their extracellular products can be demonstrated.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. No practical measures of prevention except those for Group A streptococcal infections (see Scarlet Fever, p. 179).
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: In selected endemic areas (USA); in many states and countries not a reportable disease, Class 3B

(pp. 5, 6). Areas of high incidence will profit materially by encouraging individual case report over prescribed periods sufficient to acquire epidemiological data necessary for improved methods of control.

2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: None.
8. Specific treatment: Individuals known to have had rheumatic fever or convalescent from that disease should receive chemoprophylaxis for long periods thereafter, possibly throughout life, at least until age 18 years, and past that age if necessary to give a period of 5 years from last attack. Either a sulfonamide drug or penicillin may be used, given orally throughout the year. Prior to prophylaxis, proper treatment should be instituted to free the patient of Group A streptococci (see Scarlet Fever, p. 180).

Although steroid therapy is widely used salicylates are the method of choice in management of the acute phase, preferably acetylsalicylic acid, also sodium salicylate, continued for duration of active disease. Patients must be protected from intercurrent infection, particularly Group A hemolytic streptococci.

C. Epidemic measures: Epidemics of rheumatic fever occur only in association with epidemics of Group A streptococcal infection. Proper therapy of the streptococcal infection (see Scarlet Fever, p. 180) will prevent the subsequent development of rheumatic fever and thus prevent about half of the cases of rheumatic fever.

D. International measures: None.

RICKETTSIALPOX

1. **Identification**—Rickettsialpox has an initial skin lesion, chills, fever, varicelliform rash, and a mild to severe course; even before specific therapy, fatality was less than 1%. The initial lesion is a firm red papule appearing about a week in advance of fever, most commonly on the covered parts of the body. The papule becomes vesicular, then covered by a scab, and after about 3 weeks leaves a small pigmented scar. Fever, often preceded by chills, is remittent, with peaks of 39.4° to 40.6° C (103° F to 105° F) and usually lasts less than 1 week. Headache, muscular pain, and general malaise are frequent. The secondary rash is manifest 3 to 4 days after onset of fever; has no characteristic distribution but seldom occurs on palms or soles; progresses through papular and papulovesicular stages, lasting usually less than 1 week and leaving no scars; local lymphadenopathy occurs in the region of the initial lesion, but splenomegaly or generalized lymph node enlargement is uncommon.

Specific diagnosis is by complement-fixation test, positive between the second and third week of the disease.

2. **Occurrence**—Approximately 150 cases occur annually in New York City, principally among inhabitants of apartment houses where the mouse, mite and rickettsia maintain a natural cycle of infection. A few cases

have been recognized in Boston, Hartford, Philadelphia, and Cleveland. Occurs in the USSR in the same pattern as in the USA; *R. akari* has been recovered from a field mouse in Korea but disease in man is unrecognized there.

3. **Infectious agent**—*Rickettsia akari*, a member of the spotted fever group of rickettsiae.
4. **Reservoir and source of infection**—Reservoir is the infected house mouse (*Mus musculus musculus*). Source of infection is an infective mite.
5. **Mode of transmission**—From mouse to mouse and probably from mouse to man by rodent mite, *Allodermanyssus sanguineus*.
6. **Incubation period**—Probably 10 to 24 days.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Duration of infectivity of mouse for mite, and mite for mouse or man are unknown.
8. **Susceptibility and resistance**—Susceptibility appears general. Duration of immunity after attack is unknown.
9. **Methods of control**—

A. Preventive measures:

1. Rodent and mite control by elimination of mice and mouse harborages, including proper care and firing of incinerators in dwellings, and application of residual miticides (aldrin and others) to infested areas. Commercial vaccine not available and not currently needed.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas (USA); in most states and countries not a reportable disease, Class 3B (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Search for infested mice in dwelling and, if feasible, undertake isolation of rickettsiae from rodents and mites.
8. Specific treatment: The tetracycline antibiotics and chloramphenicol are equally effective.

- C. Epidemic measures:** When groups of cases occur in the same or adjacent dwellings the preventive measures listed under 9A should be applied. Other residents should be observed and promptly treated if the disease develops.

- D. International measures:** None.

RIFT VALLEY FEVER

1. **Identification**—An acute viral infection of sheep, cattle, and other animals. Infection in man starts abruptly with fever of 38.2° C to 40.5° C (101° F to 105° F) persisting for several days. A remission of 1 to 3 days is usual, followed by a second peak of shorter duration and exacerbation of symptoms which vary greatly but commonly include nausea,

epigastric pain, sensation of fullness over the liver, and marked aversion to food. Rigor, violent headache, acute conjunctivitis and photophobia, and aching pains in the back and joints promptly follow. The usual course is 7 to 10 days, with much prostration and frequent relapse. Convalescence is prolonged over a further 4 to 6 weeks. The most distressing sequel is a central scotoma following retinal hemorrhage. Deaths are rare, in contrast to the high fatality in lambs and calves. One of the Arthropod-Borne Viral Fevers, p. 33.

Laboratory confirmation is by isolation of the virus from blood through mouse inoculation, and by test for neutralizing antibodies in early and late serums.

Two somewhat similar diseases of sheep, Wesselsbron Disease and Middleburg Disease, occur in South Africa and occasionally attack man. They are to be differentiated from Rift Valley Fever. Geographical distribution is much the same, the onset likewise abrupt, but the clinical course is more prolonged. The diseases are of viral origin and presumably transmitted by a mosquito. Control measures are as for Rift Valley Fever.

2. **Occurrence**—An uncommon and sporadic disease of man, mainly affecting males. Occurs as a naturally acquired infection only in Africa, from the Union of South Africa north to the Sudan, and in close association with epizootics among sheep and sporadic infection of cattle and goats. Laboratory infections are reported from Europe and America.
3. **Infectious agent**—The virus of Rift Valley Fever.
4. **Reservoir and source of infection**—An unidentified wild rodent is thought to be the primary reservoir. Sheep, cattle, and goats are affected along with man, epizootics usually preceding epidemics in man. Probable source of infection is an infective mosquito.
5. **Mode of transmission**—Most infections appear to be spread by bite of a mosquito. *Aedes caballus* is an efficient vector, along with *A. theileri* and *A. circumluteolus*, in cycles involving domestic animals and man, *A. caballus* primarily in high veld country and *A. circumluteolus* in low veld. Virus has been isolated in nature from at least 6 species of *Eretmapodites*, judged important in forest cycles of infection. Numerous cases in man relate to persons performing autopsies on livestock, or to butchers and housewives preparing meat. The mechanism in laboratory infections is unknown; some are concerned with autopsy of experimental animals, others may be air-borne.
6. **Incubation period**—Usually 2 days, seldom more than 3 days.
7. **Period of communicability**—During viremia of animals and man; in man until final subsidence of fever. Mosquitoes become infective after about 20 days and believed to remain so through life.
8. **Susceptibility and resistance**—Man is relatively resistant in comparison with other animals, but infection is frequent during epizootics of domestic animals. Neutralizing antibodies have been demonstrated years after recovery from laboratory infection.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Precautions in care and handling of infected animals and their products.
 2. Protection in endemic areas against mosquitoes and other possible arthropod vectors.
 3. The frequency of laboratory accidents is such as to require extreme care by those working with the virus.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; not a reportable disease in most countries, Class 3B (pp. 5, 6).
2. Isolation: None; protect patients from exposure to mosquitoes or other arthropods.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: For the individual case, of little value. A community problem. (See 9C.)
8. Specific treatment: None.

C. Epidemic measures: Not applicable to man; a sporadic disease. The problem is of domestic animals and the surrounding biologic environment. The need is for epidemiologic investigation in attempt to identify arthropod vector and natural reservoir. Vaccination of susceptible animals with an attenuated strain of virus is widely and effectively used in control of epizootics.

D. International measures: Restrict movement of animals from enzootic areas to those free from the disease.

RINGWORM

Ringworm is a general term applied to mycotic infections of keratinized areas of the body (hair, skin, and nails). Various genera and species of fungi known collectively as the dermatophytes are causative agents. For convenience in presentation, the dermatophytoses are subdivided according to sites of infection as follows: *Tinea capitis* (ringworm of the scalp), *tinea corporis* (ringworm of the body), *tinea pedis* (athlete's foot) and *tinea unguium* (ringworm of the nails). Synonyms: Favus, Jocky-Itch, Tinea, Athlete's Foot, The Dermatomycoses, The Dermatophytoses.

A. RINGWORM OF SCALP (TINEA CAPITIS)

1. Identification—Infection begins as a small papule and spreads peripherally, leaving scaly patches of alopecia (baldness). Infected hairs become brittle and break off easily. Occasionally boggy, raised, and suppurative lesions develop, called kerions. Examination of the scalp under Wood light for fluorescence is helpful in certain ringworm infections.

Microscopic examination of hairs, cleared with sodium hydroxide solution, shows arthrospores within the hair (endothrix type) or surrounding it (ectothrix type). The fungus should be cultured for genus and species identification.

Favus of the scalp is a variety of *tinea capitis* caused by *Trichophyton schoenleinii* and characterized by formation of small, yellowish, cup-like crusts or scutulae giving the appearance of being stuck on the scalp. Affected hairs do not break off but become gray and lusterless, eventually fall out and leave baldness which may be permanent. Microscopic examination of scales and hair in sodium hydroxide reveals numerous arthrospores in the scutulae; the interior of the hair is filled with long branching mycelial filaments and characteristic air spaces.

Tinea capitis is distinguished easily from Piedra, a fungus infection

of the hair occurring in South America and some countries of Southeast Asia, an infection characterized by hard "gritty" nodules on the hair shafts.

2. **Occurrence**—Ringworm of the scalp caused by *Microsporum audouinii* is widespread in the United States, particularly in urban areas. *M. canis* infection occurs both in rural and urban areas wherever infected cats and dogs are present. *Trichophyton mentagrophytes* and *T. verrucosum* (faviforme) infections are common in rural areas where the infection exists in cattle, horses, rodents, and wild animals. *T. tonsurans* infections are epidemic in urban areas in southwestern United States, Puerto Rico, and Mexico. Incidence in children is higher than adults, males are infected more frequently than females; significant differences in race have not been noted. Infection of domestic animals is more common in damp cold seasons, especially when confined to barns, and incidence of human infection with animal-transmitted strains is thus higher in colder weather.
3. **Infectious agents**—Various species of *Microsporum* and *Trichophyton*. Identification of genus and species is important epidemiologically and for determining prognosis.
4. **Reservoir and source of infection**—Reservoirs are man and infected animals, especially dogs, cats, and cattle. Sources of infection are such materials as the backs of theater seats, barber clippers, toilet articles, or clothing contaminated with hair from infected animals or man; *Microsporum gypsum* is commonly present in soil.
5. **Mode of transmission**—Direct contact with reservoirs or sources of infection.
6. **Incubation period**—10 to 14 days.
7. **Period of communicability**—As long as infected lesions are present, and as long as viable spores are present on contaminated materials.
8. **Susceptibility and resistance**—Children before the age of puberty are notoriously susceptible to *Microsporum audouinii* infection and most adults are resistant. All ages are subject to *M. canis* and *Trichophyton* infections but children are more susceptible. No permanent immunity is developed.
9. **Methods of control**—
 - A. **Preventive measures**: In epidemic areas young children should be surveyed by Wood light before entering school. The public, especially parents, should be warned of the dangers of acquiring infection from infected children as well as from dogs, cats, and other animals. Effective control of animal ringworm is essential to control of infection in man.
 - B. **Control of patient, contacts, and the immediate environment**:
 1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6). School outbreaks should be reported to school authorities.
 2. Isolation: Impractical. The patient should be under a regulated regimen of treatment with periodic visits to physician or clinic. Hair should be covered with a cap which can be sterilized frequently.
 3. Concurrent disinfection: Contaminated caps should be boiled after use.
 4. Terminal disinfection: None.
 5. Quarantine: Not practicable.

6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Household contacts, and pets and farm animals, for evidence of infection.
 8. Specific treatment: Ointments containing salicylanilid, copper undecylenate or any one of a variety of similar compounds. Epilation by Roentgen-ray is instituted if progress is not satisfactory. Examine weekly and take cultures to assure recovery is complete. Griseofulvin by mouth is treatment of choice.
- C. Epidemic measures:** Epidemics in a school or institution require special measures such as education of children and parents, and enlistment of services of physicians and nurses for diagnosis. Follow-up surveys are important.
- D. International measures:** Examination of scalps of immigrants coming from areas of high prevalence of favus (*T. schoenleinii* or *T. violaceum* infections). Infected persons should be detained, placed under treatment, and entry deferred until free of the disease, a procedure not to be applied loosely to all ringworm infection.

B. RINGWORM OF BODY (TINEA CORPORIS)

1. **Identification**—Cutaneous infections other than of the scalp, bearded areas, and feet, characteristically appearing as flat spreading ring-shaped lesions. The periphery is reddish, vesicular, or pustular, and may be dry and scaly or moist and crusted. As lesions progress peripherally, the central area often clears and skin appears normal.
Scrapings from the advancing margins, cleared in sodium hydroxide and examined microscopically show segmented branching filaments. Final identification is by culture.
2. **Occurrence**—World-wide and relatively frequent. Males are infected more than females. All ages are susceptible and racial differences are immaterial.
3. **Infectious agents**—*Epidermophyton floccosum* and various species of *Microsporum* and *Trichophyton*.
4. **Reservoir and source of infection**—Reservoir is man and animals; sources of infection are skin and scalp lesions of infected persons, lesions of animals, contaminated floors, shower stalls, benches, and similar articles.
5. **Mode of transmission**—Direct contact with reservoirs or sources of infection.
6. **Incubation period**—10 to 14 days.
7. **Period of communicability**—As long as lesions are present and viable spores are present on contaminated materials.
8. **Susceptibility and resistance**—Susceptibility is general. Clinical manifestations are commonly exaggerated under conditions of friction and excessive perspiration, as in axillary and inguinal regions.
9. **Methods of control**—
 - A. Preventive measures:** Adequate sterilization of towels and general cleanliness in showers and dressing rooms of gymnasiums, especially repeated washing of benches. A fungicidal agent such as cresol should be used for disinfection.
 - B. Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6). Case report to school

authority of infections of children involving exposed parts of the body is desirable.

2. Isolation: Infected children should be excluded from gymnasiums, swimming pools, and activities likely to lead to exposure of others.
 3. Concurrent disinfection: Of clothing in contact with infected parts of the body.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Examination of school and household contacts, and of household pets and farm animals.
 8. Specific treatment: Thorough bathing with soap and water, removal of scabs and crusts, and application of any of a number of ointments containing salicylic acid or one of the higher fatty acids (propionic acid, undecylenic acid). Griseofulvin by mouth is treatment of choice.
- C. **Epidemic measures:** Education of children and of parents concerning the nature of the infection, its mode of spread and necessity of maintaining good body hygiene.
- D. **International measures:** None.

C. RINGWORM OF FOOT (TINEA PEDIS)

1. **Identification**—Scaling or cracking of the skin, especially between the toes, or blisters containing a thin watery fluid are so characteristic that most laymen recognize "athlete's foot." In severe cases, vesicular lesions appear on various parts of the body, especially the hands. These dermatophytids do not contain the fungus and constitute an allergic reaction to fungus products.
Microscopic examination of sodium hydroxide-treated scrapings from lesions between the toes reveals segmented branching filaments.
2. **Occurrence**—World-wide and a common disease. Adults more often affected than children; males more than females. No differences in racial susceptibility. Infections are more frequent in hot weather.
3. **Infectious agents**—*Epidermophyton floccosum* and various species of *Trichophyton*.
4. **Reservoir and source of infection**—Reservoir is man; sources of infection are skin lesions of infected persons or contaminated floors, shower stalls, and other articles used by infected persons.
5. **Mode of transmission**—Direct contact with reservoirs or sources of infection.
6. **Incubation period**—Approximately 10 to 14 days.
7. **Period of communicability**—As long as infected lesions are present and as long as viable spores are present on contaminated materials.
8. **Susceptibility and resistance**—Susceptibility is variable and infection may be inapparent. Second attacks are frequent.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. As for tinea corporis above.
 2. Maintenance of strict personal hygiene, with special care in drying areas between toes after bathing.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6). School outbreaks should be reported to school authorities.
2. Isolation: None.
3. Concurrent disinfection: Socks of heavily infected individuals should be boiled to prevent reinfection. Shoes should be placed in a box and subjected to formaldehyde for several hours, followed by airing to prevent irritation of skin from residual formalin.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: None.
8. Specific treatment: Ointments may be used as recommended for *tinea corporis*. Exposure of feet to air through wearing sandals and the use of powders are often beneficial. Griseofulvin may be used in resistant cases.

C. Epidemic measures: Thorough cleaning and washing down of gymnasiums, showers, and similar sources of infection. Education of the public concerning the nature of the infection and its mode of spread.

D. International measures: None.

D. RINGWORM OF NAILS (TINEA UNGUIUM)

1. **Identification**—A chronic infection involving one or more nails of the hand or foot. The nail gradually thickens, becomes discolored and brittle, and an accumulation of caseous-appearing material forms beneath the nail, or nail becomes chalky and disintegrated.

Microscopic examination of sodium hydroxide preparations of the nail and of detritus beneath the nail shows segmented branching mycelial filaments. The diagnosis should be confirmed by culture.

2. **Occurrence**—Common; adult males more frequently infected than females. No seasonal or racial differences.
3. **Infectious agents**—*Epidermophyton floccosum* and various species of *Trichophyton*.
4. **Reservoir and source of infection**—Reservoir is man; source of infection is presumably skin or nail lesions of infected persons, possibly contaminated floors and shower stalls.
5. **Mode of transmission**—Presumably by direct extension from lesions of the foot of the patient infected. Usually no transmission even to close family associates.
6. **Incubation period**—Unknown.
7. **Period of communicability**—As long as infected lesions are present.
8. **Susceptibility and resistance**—Injury to nail predisposes to infection. Reinfection is frequent.
9. **Methods of control**—

A. Preventive measures: The measures described above for prevention of *tinea pedis*.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
2. Items 2 through 7, see *tinea pedis* above.

8. Specific treatment: Scrape off as much of affected nail as possible. Apply ointments or other medication of types described for tinea pedis. Repeat frequently until nail appears normal. Griseofulvin is treatment of choice.

C. *Epidemic measures*: Not applicable.

D. *International measures*: None.

ROCKY MOUNTAIN SPOTTED FEVER

1. **Identification**—This prototype of the diseases caused by the spotted fever group of rickettsiae is an infectious endangitis characterized by sudden onset of fever, ordinarily persisting for two weeks, headache, conjunctival injection and a maculo-papular rash. The rash appears on the extremities about the third day and spreads rapidly to most of the body, including palms and soles, before becoming petechial. Fatality is about 20% in absence of specific therapy; death is uncommon with prompt treatment.
The Weil-Felix reaction with *Proteus* OX-19 and usually with OX-2 becomes positive late in the second week; complement-fixation tests, using specific rickettsial antigen, a few days later.
2. **Occurrence**—Throughout most of the United States during spring and summer but most prevalent in Rocky Mountain and Middle Atlantic seaboard states. In western United States adult males are most affected while in the East children are more frequently attacked; infection rates are related to opportunity for contact with infected ticks, and fatality increases with age. Infection also occurs in western Canada, western and central Mexico, Colombia, and Brazil. Although limited to the Western Hemisphere, the disease is closely related to tick-borne rickettsial infections of other continents.
3. **Infectious agent**—*Rickettsia rickettsii*.
4. **Reservoir and source of infection**—Ticks, rabbits, field mice, and dogs are reservoirs. The infection is passed from generation to generation in ticks, which consequently are not only the immediate source of infection but also a reservoir. The infectious agent is probably maintained in nature by infected and noninfected larvae feeding upon susceptible wild rodents.
5. **Mode of transmission**—Ordinarily by bite of infected tick but contamination of skin with crushed tissues or feces of such tick may lead to infection in man. In eastern and southern United States the common vector is the dog tick, *Dermacentor variabilis*; in northwestern United States, the wood tick, *Dermacentor andersoni*; in southwestern United States occasionally the Lone Star tick, *Amblyomma americanum*. In Brazil, *Amblyomma cajennense* is the common vector. The rabbit tick *Haemaphysalis leporis palustris* is infected in nature but does not bite man.
6. **Incubation period**—From 3 to about 10 days.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. The tick remains infective for life, commonly as long as 18 months.
8. **Susceptibility and resistance**—Susceptibility is general. One attack confers immunity which may or may not be permanent. Suitable vaccines induce immunity.

9. Methods of control—

A. Preventive measures:

1. Personal prophylaxis by avoiding tick-infested areas when feasible, by careful removal of ticks from the person as promptly as possible without crushing, and with protection of the hands when removing ticks from animals. Tick repellents of value are diethyltoluamide and dimethylphthalate.
2. Measures for reducing tick populations are generally impractical. Clearing the land, reducing small wild mammal populations, stray dog control, and removing ticks from livestock by dipping may help. In selected areas direct application of appropriate insecticides gives excellent control of some tick vectors. DDT, chlordane, dieldrin, lindane, and benzene hexachloride are used for this purpose.
3. Vaccines containing killed *R. rickettsii* lessen the chance of infection and result in lower fatality. Since advent of specific therapy, vaccination is generally limited to persons at high risk. Reinforcing doses at yearly intervals are necessary to maintain protection.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas (USA); in many countries not a reportable disease, Class 3B (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: Destroy all ticks on patients.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Unnecessary.
7. Investigation of contacts and source of infection: Not profitable except as a community measure; see 9C.
8. Specific treatment: The tetracycline antibiotics or chloramphenicol in daily oral doses until patient is afebrile (usually 3 days) and for 1 or 2 additional days.

C. Epidemic measures: In hyperendemic areas particular attention should be paid to identification of infected ticks and of infested areas, and to recommendations in 9A1, 2.

D. International measures: None.

RUBELLA

1. Identification—A mild febrile infectious disease with a rash of variable character, sometimes resembling that of measles, sometimes that of scarlet fever, and sometimes an admixture of both; few or no constitutional symptoms but almost always enlargement of the postauricular, suboccipital, or postcervical group of lymph nodes; occasionally others. Mild catarrhal symptoms may be present. Infection without a rash has been produced experimentally. Leucopenia is usual during fever. **Synonym:** German measles.

Because of inconsistencies in clinical and epidemiological behavior, differentiation of rubella from a number of other mild viral infections of similar nature is often required:

Erythema Infectiosum (Fifth Disease): A mild nontuberculous erythematous eruption occurring in epidemics among children. Characterized

clinically by a malar flush and reddening of the skin which occurs, fades, and recurs; exaggerated by exposure to sunlight, and unaccompanied by constitutional symptoms. Outbreaks are now recognized more frequently than formerly; not clear that this represents an increased occurrence. Of no significance as far as morbidity is concerned, but must be differentiated from rubella and from other exanthemata listed here. A virus of presumed etiologic significance has been isolated from patients but needs confirmation.

Exanthem Subitum (Roseola Infantum, Sixth Disease): An acute illness with sudden onset, usually confined to children under 4 years of age and commonly those about 1 year. A sudden rise in temperature of intermittent type, sometimes to 105° F or 106° F, lasts 3 to 5 days. Lysis of fever ordinarily is followed by a typical maculopapular rash on the trunk and later on the rest of the body. The rash fades rapidly. Prevalence is greatest in spring and fall. The incubation period is about 10 days, with a range of 7 to 17 days. The disease is only mildly communicable. Many inapparent and unrecognized infections seemingly occur among older children. An infectious agent has been isolated but confirmation is awaited.

Other Infections: Macular and maculopapular rashes occur in about 20% of patients with infectious mononucleosis (Glandular Fever, p. 117); also observed in infections with viruses of the ECHO group (Types 2, 4, 6, 9, 14, 16), most commonly in ECHO 9 outbreaks and usually in association with meningeal reaction, fever to 104° F, headache, sore throat, and gastroenteritis. Boston Exanthem is identical with ECHO Type 16 infection. Similar rashes occasionally are associated with Group A Coxsackie infections. Presence of Koplik spots and acute respiratory symptoms suggest measles.

2. **Occurrence**—Epidemic in expression, mostly in childhood, but with more adult patients than in measles. Appreciable epidemics are recorded in military practice and among college students, more prevalent in winter and spring than in other seasons. World-wide distribution and a common communicable disease.
3. **Infectious agent**—The virus of rubella.
4. **Reservoir and source of infection**—Reservoir is man; source of infection is nasopharyngeal secretions of infected persons.
5. **Mode of transmission**—By droplet spread or direct contact with patient, or by indirect contact with articles freshly soiled with discharges from nose or throat. Air-borne transmission also occurs.
6. **Incubation period**—From 14 to 21 days; usually 18 days.
7. **Period of communicability**—For at least 4 days after onset of catarrhal symptoms and probably not much longer, the exact period being undetermined. Highly communicable.
8. **Susceptibility and resistance**—Susceptibility is general among young children. One attack usually confers permanent immunity.
9. **Methods of control**—Efforts to control rubella are prompted by the hazard of significant congenital defects in offspring of women who acquire the disease during pregnancy; most commonly congenital cataracts, heart disease and deaf mutism. The extent of the risk is variously described, but approximately 10% of living infants born after maternal rubella during the first trimester of pregnancy have anomalies.

A. Preventive measures:

1. No attempt should be made to protect female children in good health against exposure to the disease before puberty. Deliberate exposure has been recommended by some authorities.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory report of epidemics; case report ordinarily serves no useful purpose; may be required specifically where contacts include susceptible women in first 4 months of pregnancy, Class 4 (p. 6).
2. Isolation: None, except where contacts include a woman in early pregnancy; then under direction of the attending physician for 5 days after onset.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Immune serum globulin (gamma globulin) has provided irregular protection against rubella. Its use should be considered only for adult female contacts with no history of rubella who are within the first four months of pregnancy. Large doses are necessary, at least 20 ml intramuscularly.
7. Investigation of contacts and source of infection: Of no practical value except to clarify possible confusion with scarlet fever; and to identify adult female contacts in the first 4 months of pregnancy.
8. Specific treatment: None.

C. Epidemic measures: None.**D. International measures: None.**

SALMONELLOSIS

Salmonella infections include a variety of clinical syndromes; the three main types are enteric fever, acute gastroenteritis, and septicemia. Every *Salmonella* strain is potentially capable of producing any of the main clinical types. A Salmonella infection also may begin as an acute gastroenteritis and develop into an enteric fever or septicemia. The infectious agent may localize in any tissue of the body, producing abscesses and causing arthritis, cholecystitis, endocarditis, meningitis, pericarditis, pneumonia or pyelonephritis.

The commonest clinical manifestation of Salmonella infection is an acute gastroenteritis, a form of food infection (see also Staphylococcal Food Poisoning, p. 80, and Botulism, p. 44) to which attention is here limited.

Salmonella Gastroenteritis

1. **Identification**—A common, acute intestinal infection with diarrhea and abdominal cramps. Fever, nausea, and vomiting are often present. Deaths are uncommon but somewhat more frequent than for Staphylococcal Food Poisoning. Synonym: Salmonella Food Poisoning.
Salmonella may be recovered from feces or from the site of a localized infection during the acute illness, but isolation is progressively difficult during convalescence.
2. **Occurrence**—World-wide and a common disease. Usually recognized in its epidemic form by groupings of cases among individuals having a common food supply. In such circumstances a high proportion of individuals develop clinical symptoms. Sporadic cases are frequent and range from mild to severe, the mild cases often recognized with difficulty.

3. **Infectious agents**—Numerous species of *Salmonella* of that group pathogenic for both animals and man. Primary human pathogens are excluded (see Typhoid and Paratyphoid Fevers, pp. 209, 123), also those producing disease only in animals. The more common species in the United States are *S. typhimurium*, *S. choleraesuis*, *S. newport*, *S. oranienburg*, *S. montevideo*, *S. panama*, and *S. anatum*; much variation from country to country. On a world basis *S. typhimurium* is the most common. Total serological types involved in food poisoning approximate 500.
4. **Reservoir and source of infection**—Reservoir is man; patients and convalescent carriers, especially mild and unrecognized cases; also domestic and wild animals. Source of infection is feces of infected persons and of domestic and wild animals, also eggs and dried egg powder.
5. **Mode of transmission**—Epidemics are usually traced to (1) improperly prepared food, especially meat pies and roast fowl; (2) insufficiently cooked foods containing dried or frozen hen or duck eggs; (3) unpasteurized milk or dairy products; (4) pastries contaminated by rodent feces, possibly through medium of cockroaches; (5) food prepared by an infected food handler. Sporadic cases often originate through direct contact with an infected person or animal.
6. **Incubation period**—In epidemics, 6 to 48 hours, usually about 12 hours. Unknown for sporadic cases but believed to be from 1 to 7 days.
7. **Period of communicability**—Throughout infection. Extremely variable, usually 3 days to 3 weeks or as long as carrier state persists.
8. **Susceptibility and resistance**—Susceptibility is general, with an apparently important relationship to size of infecting dose. No active or passive artificial immunization. Some species-specific immunity probably follows recovery.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Thorough cooking of all foodstuffs derived from animal sources. Particular attention to preparation of fowl, egg products, and meat dishes.
 2. Protection of prepared food against rodent or insect contamination.
 3. Refrigeration of prepared foods during storage before use.
 4. Attempt to control *Salmonella* infection among domestic animals.
 5. Meat and poultry inspection with adequate supervision of abattoirs and butcher shops.
 6. Adequate cooking or heat treatment, and protection of prepared animal feeds (meat meal, bone meal, and others) against contamination by *Salmonella*.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Obligatory report of epidemics; also suspected or confirmed cases with evidence of grouping or association with others in time and place, Class 4 (p. 6).
 2. Isolation: Exclusion of infected persons from food handling and occupations involving care of young children until cultures of feces are free from *Salmonella*.
 3. Concurrent disinfection: Of feces and of articles soiled therewith. In communities with modern and adequate sewage disposal systems, feces can be disposed of directly into sewer without preliminary disinfection.

4. Terminal disinfection: Cleaning.
5. Quarantine: Family contacts should not be employed as food handlers during period of contact nor before repeated negative feces cultures have been obtained.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Search for unrecognized mild cases and convalescent carriers among contacts.
8. Specific treatment: None; chloramphenicol and the tetracycline antibiotics have limited and irregular effect.

C. Epidemic measures:

1. Intensive search for case or carrier acting as source of infection.
2. Search for food which may have served as a vehicle of spread; attempt to determine method of contamination; culture food bacteriologically.
3. Destruction of any remainder of suspected food after samples have been taken for laboratory examination.

D. *International measures:* An International Salmonella Center in Copenhagen, Denmark, and national salmonella centers in various countries (e.g., Communicable Disease Center, Atlanta, Ga., USA, and Salmonella Reference Laboratory, Colindale, London) facilitate proper identification of the various *Salmonella* types. These centers provide uniformity of method and contribute to proper epidemiologic investigation of salmonellosis.

SCABIES

1. **Identification**—An infection caused by a crab-shaped mite whose penetrations into the skin are visible as papules and vesicles housing males and nymphs, or as tiny linear burrows containing females and their eggs. Lesions are prominent around finger webs, anterior surfaces of wrists and elbows, anterior axillary folds, belt line, thighs, and external genitalia in men; nipples, abdomen, and lower portion of buttocks in women. Itching is intense, especially at night, but complications are few except as lesions become secondarily infected by scratching.

Signs and symptoms are characteristic; diagnosis may be established by locating the female in its burrow and identifying it microscopically.

2. **Occurrence**—Widespread during time of war, poverty, or social upheaval. In stable times when soap and facilities for bathing are ample, the infection practically disappears.
3. **Infectious agent**—*Sarcoptes scabiei*.
4. **Reservoir and source of infection**—Reservoir is man; *Sarcoptes* of animals can live on man but do not reproduce in his skin.
5. **Mode of transmission**—Transfer of parasites is by direct contact and to a limited extent from undergarments or soiled sheets freshly contaminated by infected persons. The disease is frequently of venereal origin.
6. **Incubation period**—Several days or even weeks may elapse before itching is noticed.
7. **Period of communicability**—Until mites and eggs are destroyed by treatment. This should follow promptly after 1 or occasionally 2 courses of treatment a week apart.

8. **Susceptibility and resistance**—Anyone may become infected or reinfected.

9. **Methods of control**—

A. *Preventive measures:*

1. Cleanliness of person, garments, and bedclothes.

B. *Control of patient, contacts, and the immediate environment:*

1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6).

2. Isolation: Less important than treatment, education, and identification of infected contacts. Exclusion of infected children from school until treated adequately.

3. Concurrent disinfection: Proper laundering of underwear, sheets, and occasionally of blankets is indicated.

4. Terminal disinfection: Unnecessary.

5. Quarantine: None.

6. Immunization of contacts: None.

7. Investigation of contacts and source of infection: Search is made for unreported or unrecognized cases among companions or household members. Single infections in a family without spread to others are uncommon.

8. Specific treatment: A bath followed by application of 20–25% emulsion of benzyl benzoate or 1% benzene hexachloride ointment (Kwell®) to the whole body. Medication is usually applied with a brush with particular attention to infected areas. The following day a cleansing bath is taken and a change made to fresh clothing and bed linen. Itching may persist for days and is not to be regarded as a sign of superinfection; this is important, for over-treatment is common. In perhaps 5% of cases a second course of treatment is necessary after an interval of 7 to 10 days. Five % sulfur ointment is a time-honored effective treatment now little used; the regimen is similar to that just given.

C. *Epidemic measures:*

1. Treatments are undertaken on a coordinated mass basis.

2. Case-finding efforts are redoubled in order to screen whole families, military units, or suspect institutions for infected persons.

3. Soap and facilities for mass bathing and laundering are essential.

4. Education as well as treatment of infected persons and others at risk is indicated and the cooperation of civilian or military authorities, often both, is needed.

D. *International measures:* None.

SCHISTOSOMIASIS

1. **Identification**—A blood-fluke (trematode) disease with adult male and female worms living in veins of the host (mainly mesenteric, portal, and pelvic veins). Eggs there deposited produce minute granulomata and scars in organs where they lodge. Symptomatology is related to the life cycle of the parasite; *S. mansoni* and *S. japonicum* give rise primarily to intestinal and *S. haematobium* to urinary manifestations. Synonym: Bilharziasis.

2. **Occurrence**—*S. mansoni* occurs in Africa, the Arabian peninsula, north-eastern and eastern South America and the Caribbean area. *S. haematobium* occurs in Africa, the Middle East, Portugal, and a small focus in India. *S. japonicum* occurs in the Orient (China, Japan, Philippines, Celebes). No species is indigenous to continental North America. In some endemic areas more than half of the population is infected.

The larvae of certain other schistosomes of birds and rodents may penetrate the human skin causing a dermatitis known as "Swimmer's Itch." This is prevalent among bathers in lakes in many parts of the world including North America; also in certain coastal sea water beaches. These schistosomes do not mature in man.

3. **Infectious agents**—*Schistosoma mansoni*, *S. haematobium*, and *S. japonicum*.

4. **Reservoir and source of infection**—Man is the principal reservoir of both *S. haematobium* and *S. mansoni*. Primates have been found naturally infected with both species. Dogs, pigs, cattle, water buffalo, horses, field mice, and wild rats are animal hosts of *S. japonicum*. Immediate source of infection is water contaminated with larval forms (cercariae) derived from snails.

5. **Mode of transmission**—The eggs of *S. haematobium* leave the body mainly with urine; those of *S. mansoni* and *S. japonicum* with feces. The egg hatches in water and the liberated larva or miracidium enters a suitable fresh-water snail host. Free swimming larvae, cercariae, emerge from the snail after several weeks and penetrate the human skin, usually while the person is working, swimming, or wading in water; they enter the blood stream, are carried to blood vessels of the liver, develop to maturity, and then migrate to veins of the abdominal cavity. Adults of *S. mansoni* and *S. japonicum* usually remain in mesenteric veins; those of *S. haematobium* usually migrate through anastomoses into the pelvic veins. Eggs are deposited in venules and by necrosis of tissue escape into the lumen of bowel or bladder, but may lodge in other organs.

6. **Incubation period**—Systemic manifestations usually begin when the worms are reaching maturity, about 4 to 6 weeks after infection. Eggs usually are found in feces or urine a week or two after onset of symptoms.

7. **Period of communicability**—As long as eggs are discharged in urine or in feces of infected persons, which may be 25 years or longer. Infected snails may give off cercariae for several months.

8. **Susceptibility and resistance**—Susceptibility is universal; whether or not resistance develops as a result of infection is controversial.

9. **Methods of control**—

A. Preventive measures:

1. Disposal of feces and urine so that eggs will not reach bodies of fresh water containing snail intermediate host. Control of animals infected with *S. japonicum*.
2. Improved irrigation and agricultural practices; drainage and reclamation of swamps.
3. Treatment of snail breeding places with molluscicides, copper sulfate, or sodium pentachlorophenate.
4. Provision of water for drinking, bathing, and washing clothes from sources free from cercariae.

5. Provision of cercaria-repellent or protective clothing against cercariae for persons required to enter contaminated water.
 6. Education of people in endemic areas regarding mode of transmission and methods of protection.
 7. Mass treatment of infected persons in endemic areas may help to reduce transmission through lessened severity and duration of the disease; in the past has not materially reduced prevalence.
- B. Control of patient, contacts, and the immediate environment:**
1. Report to local health authority: In selected endemic areas; in many countries not a reportable disease, Class 3C (pp. 5, 6).
 2. Isolation: None.
 3. Concurrent disinfection: Sanitary disposal of feces and urine.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Examine contacts for infection from a common source. The search for the source is a community effort, see 9C.
 8. Specific treatment: For *S. mansoni* and *S. haematobium*, fuadin® intramuscularly. For *S. haematobium*, miracil D (nilodin) is efficacious in light infections, less so in heavy infections; disagreeable side effects but no serious toxicity. For *S. japonicum*, tartar emetic intravenously; toxic side effects occur; also used for other species.
- C. Epidemic measures:** In areas of high incidence, or in endemic areas where outside groups such as military forces become infected, snail breeding places should be carefully determined and treated with molluscicides; entering infected water should be prohibited. Clean water should be provided, population should be examined for infection, and infected persons treated.
- D. International measures:** None.
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SCRUB TYPHUS

1. **Identification**—A rickettsial disease characterized by a primary lesion followed by a rash late in the first week of fever. The primary lesion, usually on a protected area of skin and representing the site of attachment of an infected mite, precedes the acute febrile onset by several days. Headache, conjunctival injection, and lymphadenopathy accompany the fever. A dull red maculopapular eruption appears on the trunk, extends to the extremities, and disappears in a few days. Cough and roentgenographic evidence of pneumonitis are common. In the absence of specific antibiotic therapy, fever lasts for 14 days. Fatality in untreated cases varies with locality from 1 to 40%; regularly higher among older persons. Synonyms: Tsutsugamushi Disease, Mite-Borne Typhus Fever.

Isolation of the infectious agent by inoculation of mice, and specific complement-fixation tests supplement the Weil-Felix reaction (*Proteus* OXK) in laboratory diagnosis of the disease.

2. **Occurrence**—Scrub typhus occurs in eastern and southeastern Asia, northern Australia, the Indian subcontinent, and adjacent islands. It is a place disease acquired by man in one of the innumerable small

sharply delimited "typhus islands" where the rickettsiae, the vector, and the rodent reservoir exist simultaneously. Occupational habits greatly influence sex distribution, but with few exceptions the disease is restricted to adult workers who frequent scrub or overgrown terrain. In the Pescadores Islands children are more often attacked because infected rodents and mites inhabit rock walls around gardens of homes. Epidemics occur when susceptibles are brought into endemic areas, especially in military operations, where 20 to 50% of men have been infected within weeks or months.

3. **Infectious agent**—*Rickettsia tsutsugamushi*.

4. **Reservoir and source of infection**—Mites and various wild rodents constitute the reservoir of infection. Infected larval mites of *Trombicula akamushi* and related species which vary with locality, are the immediate source of infection. Infection is maintained additionally by a mite-wild rodent-mite cycle.

5. **Mode of transmission**—By the bite of infected larval mites; nymphs and adults do not feed on vertebrate hosts.

6. **Incubation period**—Usually 10 to 12 days but varies from 6 to 21.

7. **Period of communicability**—Under natural conditions not directly transmissible from man to man.

8. **Susceptibility and resistance**—Susceptibility is general. An attack confers prolonged immunity against the homologous strain of *R. tsutsugamushi* but only transient immunity against heterologous strains. Heterologous infection within a few months results in mild disease but such infection after a year produces the typical illness. Second and even third attacks of naturally acquired scrub typhus occur among persons who spend their lives in endemic areas. Inactivated vaccine is useless as an immunizing agent.

9. Methods of control—

A. Preventive measures:

1. The aim is to prevent contact with infected mites, to eliminate mites and rodents from particular sites, and to promote resistance to the disease.
2. Personal prophylaxis against the mite vector is by use of clothes and blankets impregnated with miticidal chemicals, such as benzyl benzoate, together with application of mite repellents, N-diethyl-toluamide, to exposed skin surfaces.
3. In military practice selected camp sites are cleared of vegetation by stripping with a bulldozer, the vegetation destroyed by burning, the area sprayed with residual miticidal chemicals (chlordane, dieldrin effective for several weeks or a month), and rodent control measures instituted.
4. Attempts to render man insusceptible to scrub typhus are generally impractical.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas, with clear distinction from endemic and epidemic typhus; in many countries not a reportable disease, Class 3A (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.

6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: None.
8. Specific treatment: One of the tetracycline antibiotics or chloramphenicol orally in a loading dose followed by divided doses daily until patient is afebrile (average 30 hours). If treatment is instituted within the first 3 days a second course should be given about the eighth day after onset to prevent relapse.

C. Epidemic measures:

1. Rigorously employed procedures described in 9A2 and 9A3 for all persons in the affected area.
2. Daily observation for fever and appearance of primary lesion of all persons at risk; institute treatment promptly at first indication of illness.
3. Consider use of chemoprophylaxis for key personnel.

D. International measures: None.

SMALLPOX

1. **Identification**—An exanthematous disease characterized by sudden onset with fever, chills, headache, severe backache, and prostration, continuing for 3 to 4 days. The temperature then falls and a rash appears which passes through stages of macule, papule, vesicle, and pustule, forms crusts and finally scabs which fall off at about the end of the third week. The eruption is usually symmetrical and general, more profuse on prominences, extensor surfaces, flexures and depressions. Most abundant and earliest on the face, next on forearms, wrists and hands, and favoring the limbs more than the trunk, especially distally. More abundant on shoulders and chest than on loins or abdomen, but lesions may be so few as to be overlooked.

Variola major (classical smallpox) is a severe disease which in recent years has been running true to type with fatality about 30%. An uncommon fulminating form, hemorrhagic smallpox, is characterized by purpura, hemorrhages into the skin, and death within 3 to 4 days, usually before the typical rash appears. Variola minor (alastrim) is a milder form of the disease which in recent years also has run true to type with fatality less than 1%. It has mild prodromal symptoms, a discrete and scanty rash, and more rapid progression of lesions.

Modification of both the mild and classical forms of the disease, in timing and maturation of rash and in other clinical features, is to be expected in persons with waning immunity after vaccination.

Laboratory confirmation is by isolation and identification of virus, using embryonated eggs inoculated with vesicular or pustular fluid. More rapid diagnosis is sometimes possible by using human cutaneous lesions as antigen in complement-fixation test with rabbit antiviral serum.

2. **Occurrence**—Distributions within countries of the world range from sporadic to endemic to epidemic, varying widely according to immunity status of a population and frequency of imported infection. Incidence is greatest in winter and least in summer. The disease is still a serious problem in parts of Asia, Africa, and South America.
3. **Infectious agent**—The virus of smallpox.

4. **Reservoir and source of infection**—Reservoir is man; source of infection is respiratory discharges of patients and lesions of skin and mucous membrane, or materials contaminated therewith.
5. **Mode of transmission**—By contact with persons sick with the disease. Contact need not be intimate; aerial transmission may occur over short distances within closed spaces. Also spread by articles or persons freshly contaminated by respiratory discharges or by material from lesions of the skin and mucous membranes of the sick; scabs remain infectious for variable periods.
6. **Incubation period**—From 7 to 16 days, commonly 12 days. A good working rule in epidemiological investigations is 10 days from rash to onset of second illness, or 14 days from rash to rash, the latter being the more useful in field practice.
7. **Period of communicability**—From first symptoms to disappearance of all scabs and crusts, a period of about 2 to 3 weeks. Most communicable in the early stages of the disease.
8. **Susceptibility and resistance**—Susceptibility is universal, although exposure of a susceptible person does not always result in the disease. Permanent immunity usually follows recovery; second attacks are rare. Immunity acquired by vaccination gradually diminishes.
9. **Methods of control**—

A. Preventive measures:

1. Vaccination at about the third month of age, revaccination on entering school, and of all persons facing unusual exposure as in travel to endemic regions or presence of smallpox. Revaccination under conditions of sustained high risk may be practiced at intervals as short as every 6 months, as with troops in military operations.

Workers in hospitals, including physicians, nurses, attendants, laboratory and laundry employees, should as a group maintain their immune status through prompt vaccination upon employment and revaccination every 3 years. Such persons often have been responsible for spread of newly introduced infection in communities with little recent smallpox.
2. Measures to assure available supplies of potent glycerinated smallpox vaccine maintained below freezing up to the hour of vaccination, to include time in transit or shipment. Dried calf lymph vaccine should be used where facilities for transportation and refrigeration of vaccine are poor, mainly in the tropics.
3. To avoid complications, insertion of vaccine should be over a small area of skin, not more than one-eighth inch in any direction, and preferably by multiple pressure method or by acupuncture. If the scratch method is employed, care should be taken to avoid drawing blood. The site should be kept dry and cool without the use of shields or dressings. The deltoid area of the arm is the ordinary site; leg vaccination should be avoided. Eczema, agammaglobulinemia and leukemia are contraindications against vaccination; patients with eczema should not come in contact with recently vaccinated persons.

Revaccination with a fully potent vaccine, of persons immunized more than 10 years previously, gives at least 50% of vaccinoid (accelerated) reactions. The site of revaccination should be carefully observed and the reaction recorded twice, on the third and ninth days after vaccination, to determine whether the maximum

diameter of redness occurred in less than 3 days (immediate or early reaction, formerly called immune reaction), between 3 and 7 days (vaccinoid reaction), or after 7 days (vaccinia). The common result is either an immediate or accelerated reaction. The accelerated reaction indicates persisting protection; the immediate reaction, if marked by a firm indurated papule at the site of revaccination, probably does. Errors in interpretation of the early reaction are frequent; inactive vaccine sometimes gives the same response, trauma also confuses and sometimes secondary infection. If there is any doubt, revaccinate; with no reaction, always revaccinate.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report universally required by international regulation, Class 1 (p. 5).
2. Isolation: Hospital isolation in screened wards or rooms until all scabs and crusts have disappeared.
3. Concurrent disinfection: Oral and nasal discharges to be deposited in a paper bag or other suitable container and burned. All articles associated with the patient to be sterilized by high pressure steam or by boiling.
4. Terminal disinfection: Thorough cleaning of sick room and furniture; sterilization of mattress, pillow, and bedding.
5. Quarantine: All persons living or working on the same premises as the person who develops smallpox, or otherwise having intimate exposure, should be considered contacts, and promptly vaccinated or revaccinated, or quarantined for 16 days from last exposure. If such contacts are considered immune by reasons of prior attack or successful revaccination within the previous 3 years, they should be kept under surveillance until the height of the reaction to the recent vaccination has passed. If the contact is not considered immune, he should be kept under surveillance until 16 days have passed since last contact. Any rise of temperature during surveillance calls for prompt isolation until smallpox can be excluded.
6. Immunization of contacts: Prompt vaccination with a potent vaccine of all contacts, casual as well as intimate.
7. Investigation of contacts and source of infection: The immediately prior case should be sought assiduously. Adults with chickenpox or patients with hemorrhagic or pustular lesions of the skin, particularly those associated in time or place with known smallpox, need careful review for errors in diagnosis.
8. Specific treatment: None.

C. Epidemic measures:

1. Hospital care of patients and suspects until no longer infective.
2. Careful listing of all contacts and rigorous enforcement of quarantine until protected by successful vaccination; surveillance for 16 days from last exposure.
3. Immediate publicity by all available means, giving a simple, clear, and frank statement of the situation and urging all individuals in the area to be vaccinated. Provide potent vaccine to physicians and hospitals; establish vaccination clinics for persons without a private physician.
4. Mass immunization of whole populations of a community or larger area is an emergency measure to be used when smallpox has entered a community and given evidence of material spread.

D. International measures:

1. Telegraphic notification of WHO and adjacent countries by governments of a first case of smallpox arising within the country. In special circumstances and in some countries variations are made in these requirements to include place and number.
2. Measures applicable to ships, aircraft, and land transport arriving from smallpox areas are specified in International Sanitary Regulations, World Health Organization, annotated edition, Geneva, 1957.
3. International travelers: Infected persons or suspects are not permitted to depart a country. Many countries require arriving travelers to present evidence of a recent vaccination or of a previous attack of smallpox. The validity of an international certificate of vaccination against smallpox extends for a period of 3 years beginning 8 days after a successful primary vaccination, or in the event of a revaccination on the date of that revaccination.

SPOROTRICHOSIS

1. **Identification**—In commonest form a localized fungus infection of the skin, beginning as a nodule which ultimately forms a necrotic ulcer. As the nodule grows, lymphatics draining the area gradually become firm and cordlike, forming a series of nodules which in turn soften and ulcerate. Other forms of sporotrichosis such as skeletal, visceral, and disseminated infection are rare in the United States. A fatal result is uncommon.

Laboratory confirmation is through cultivation of the fungus, rarely through observation in direct smear.

2. **Occurrence**—Cases have been reported from all parts of the world, males more frequently than females, and adults more than children; reportedly an occupational disease of horticulturists. No differences in racial susceptibility. The disease is characteristically sporadic, and relatively uncommon. An epidemic among gold miners in South Africa involved some 3,000 persons; fungus was growing on mine timbers. Occurs also in horses.
3. **Infectious agent**—*Sporotrichum schenckii*, a dimorphic fungus.
4. **Reservoir and source of infection**—Reservoir is soil, vegetation, wood; source of infection is the same, also spore-laden dust.
5. **Mode of transmission**—Introduction of fungus through skin following pricks by thorns or barbs, by slivers from wood or lumber, or through skin lesions of persons handling contaminated dressings of patients. Transmission by inhalation of spores is rare.
6. **Incubation period**—The lymphatic form may develop 3 weeks to 3 months after injury.
7. **Period of communicability**—Contamination of environment presumably for duration of active lesions. Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—Man probably is highly susceptible.
9. **Methods of control**—
 - A. **Preventive measures**: Treatment of lumber with fungicides in industries where disease occurs.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Official report ordinarily not justifiable, Class 5 (p. 6).
2. Isolation: None.
3. Concurrent disinfection: Of discharges and dressings.
4. Terminal disinfection: Thorough cleaning.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Not profitable.
8. Specific treatment: Iodides are effective; amphotericin B should be tried.

C. Epidemic measures: In the South African epidemic, mine timbers were sprayed with a mixture of zinc sulphate and triolith. This and other sanitary measures controlled the epidemic.

D. International measures: None.

STAPHYLOCOCCAL INFECTION

Staphylococci are ubiquitous. The infections they cause have been identified for years with such clinical conditions as boils, carbuncles, osteomyelitis, enterocolitis, meningitis, and numerous others. In the last decade staphylococcal infections have attained new importance and become serious problems principally for the newborn, their mothers, postoperative patients, and the debilitated at any age.

Several factors seemingly account for the change in character of staphylococcal infections: (1) *Antibiotic resistance*—Staphylococci have shown decided resistance to many known chemotherapeutic agents; excessive use of antibiotics in prophylaxis and treatment has contributed to this along with relaxed observance of aseptic technics. (2) *Particular strains* have been identified as "epidemic or hospital strains," such as 80/81. They are apparently 3 to 4 times more resistant to antibiotics than staphylococcal strains present in the community at large. (3) *Reservoir*—Medical, nursing, and ancillary medical personnel are known to harbor the so-called "epidemic strains" more frequently than persons in the general community. (4) *Hospital admissions* have increased greatly; nearly 3 times more mothers are admitted to obstetrical services than formerly. The hospital is a public gathering place (5) *Age change*—The general population now has greater numbers of aged and debilitated persons, a circumstance arising in part from the material successes of antibiotic therapy.

Staphylococcal Food Poisoning, an intoxication and not an infection, has separate consideration on page 80. The attention here is to infections, local and general.

A. IMPETIGO CONTAGIOSA

1. Identification—A purulent dermatitis, characterized initially by vesicular lesions which later become crusted seropurulent plaques, commonly of the face and hands but sometimes widely over the body; a disfiguring and offensive disease, but rarely serious.

Impetigo of the newborn (pemphigus neonatorum) is an acute vesicular or bullous eruption of the skin of newborn infants, particularly infants in nurseries. Onset is commonly between the fourth and tenth day of life as vesicles or bullae, usually first seen on the lower abdomen

but spreading to the skin of any part of the body. Vesicles rupture, thin crusts form and new lesions develop, tending to spread peripherally and often coalescing. Constitutional symptoms are unusual, but if lesions are widespread, fever, diarrhea, pneumonia, meningitis, bacteremia and death may occur.

2. **Occurrence**—Common among children, especially in warm weather. Occurs sporadically and as small epidemics in nurseries for infants, institutions for children, and summer camps. Likely to spread rapidly where personal hygiene is neglected. Incidence is high in countries such as the United States, where proportion of deliveries in hospitals is great and newborn infants are housed in nurseries.
3. **Infectious agents**—*Staphylococcus aureus*; certain specific strains often are associated with epidemics; hemolytic streptococci alone or in combination with staphylococci may be responsible for some cases.
4. **Reservoir and source of infection**—The reservoir is man; in hospitals infected infants, attendants, or visitors. Source of infection is lesions of infected persons and discharges from nose and throat.
5. **Mode of transmission**—By direct contact with moist discharges of skin lesions or indirect contact with articles recently soiled with discharges; also, contact with others whose skin or nasopharynx is contaminated or infected. Autoinoculation by scratching; some evidence of air-borne transmission through dust in hospital wards.
6. **Incubation period**—From 2 to 5 days, occasionally longer.
7. **Period of communicability**—Until lesions have healed; usually about 1 or 2 weeks. Asymptomatic carriers may disseminate the organism for months.
8. **Susceptibility and resistance**—Susceptibility general, especially among newborn infants and debilitated persons. Immunity does not follow an attack.
9. **Methods of control**—
 - A. **Preventive measures**:
 1. For hospital situations:
 - a. Scrupulous attention to aseptic precautions.
 - b. Consideration of all control measures through a hospital Infections Control Committee.
 2. For nonhospital situations:
 - a. Personal cleanliness, particularly avoidance of common use of toilet articles.
 - b. Prompt treatment of the first case in a group of children.
 - c. Prompt treatment of scabies and pediculosis.
 - B. **Control of patient, contacts, and the immediate environment**:
 1. Report to local health authority: Obligatory report of epidemics especially in institutions, schools, hospitals, and other groups of children. No individual case report, Class 4 (p. 61). A single case in a hospital or other nursery is to be interpreted as a potential epidemic; report two or more concurrent cases.
 2. Isolation: Prompt isolation of infected infants; prevent contact of infected child with other children or debilitated persons until lesions heal.
 3. Concurrent disinfection: Of dressings and moist discharges; disinfect clothes, towels, and sheets before laundering; avoid reinfection from

contaminated wash cloths, combs, and other toilet articles used by children.

4. Terminal disinfection: Wash nursery cribs and other furniture with hexachlorophene detergent after all infants discharged; boil instruments and basins, disinfect mattresses and sterilize bed clothing. Thorough cleaning of towels, combs, hair brushes, and other toilet articles.
5. Quarantine: Of all exposed newborn infants in presence of epidemic. Quarantine of children, none.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Infants: Examine nurses and attendants for presence of skin lesions; exclude those with lesions until healed. Children: In family cases of little profit.
8. Specific treatment: For infants, use appropriate antibiotic systematically. For children, remove crusts with bacitracin ointment or 5% ammoniated mercury ointment; continue either with ointment or 5% ammoniated mercury in calamine lotion; chlortetracycline ointment is also effective. For severe spreading infection use parenteral or oral penicillin, but never penicillin locally; substitute chlortetracycline or other broad spectrum antibiotics if penicillin-resistant.

C. Epidemic measures:

1. If an outbreak (more than 1 case) occurs in a nursery, isolate all patients and quarantine contacts until all have been discharged. Permit no new admissions to quarantined nursery.
2. Exclude nurses and attendants with skin infections, also asymptomatic carriers of the epidemic strains, particularly nasal carriers.
3. Investigate adequacy of nursing procedures; emphasize cleanliness and personal hygiene.
4. Nursing staffs assigned to isolation and quarantined nurseries should not work in nurseries housing normal newborn infants.

D. International measures: None.

B. GENERAL STAPHYLOCOCCAL INFECTIONS OFTEN HOSPITAL-ACQUIRED

1. **Identification**—Common, acute, and sometimes chronic bacterial infection characterized generally by suppuration affecting various body sites and systems and exhibiting a variety of lesions ranging from a solitary pustule to a fatal septicemia. Clinical manifestations vary with extent and severity of infection but commonly include malaise, fever, headache, and symptoms relating to system or organ involved.

The various manifestations of staphylococcal infection may appear at any age, but certain diseases predominate at particular ages, as follows: (1) Newborn and infants under 2 years: Impetigo, conjunctivitis, enteritis, pneumonia, septicemia, abscess of soft tissues; (2) young children: impetigo, pneumonia, empyema; (3) older children and adults: pimples, boils, carbuncles, cellulitis, pneumonia, mastitis, secondary infection of surgical and traumatic wounds; (4) elderly individuals and debilitated persons of any age with chronic disease: enterocolitis, septicemia, abscess of soft tissues and organs, and less commonly, endocarditis, pneumonia, nephritis, meningitis, osteomyelitis, and arthritis.

Verification of diagnosis depends upon isolation of a coagulase positive staphylococcus.

2. **Occurrence**—World-wide in distribution. The current problem is mainly of hospitals, fostered by laxity in aseptic technics because of the ready availability of antibiotics, and in turn exaggerated by the development of antibiotic-resistant strains of the infectious agent. Attack rates are frequently high and epidemics occur. Endemic levels in the general population have shown no appreciable increase; the potential exists for community infections through increasing numbers of discharged hospital patients with consequent introduction of antibiotic-resistant strains.
3. **Infectious agent**—Various phage types of coagulase-positive *Micrococcus pyogenes* var. *aureus*, (*Staphylococcus aureus*).
4. **Reservoir and source of infection**—Reservoir is infected persons. Source of infection is discharges from lesions of skin and from the nasopharynx; also the immediate environment of infected persons, as air, bed clothing, and sickroom articles and furniture.
5. **Mode of transmission**—Contact with patient or carrier, with articles contaminated by such persons or by droplet spread; many instances of autoinfection.
6. **Incubation period**—Variable and indefinite. For infection of the newborn from 2 to 20 days, with 4 to 6 days usual.
7. **Period of communicability**—As long as lesions are unhealed and carrier state persists.
8. **Susceptibility and resistance**—Although the human host possesses a fair degree of resistance, susceptibility is general, particularly for the newborn, infants, the elderly, and debilitated persons at any age.

9. **Methods of control**—

A. Preventive measures:

1. Strictly enforced aseptic technics in general and special hospitals, coordinated through a hospital Infections Control Committee.
2. Rooming-in of baby with mother is practiced in some hospitals.
3. Selected epidemiologic studies of patients admitted to hospital, and of patients discharged including families to which they return, especially newborns and their mothers. Examine hospital populations in order to determine prevailing local distribution of phage types, and mode and extent of transmissions.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authorities: Obligatory report of epidemics: 2 or more concurrent cases on same hospital service or ward are to be regarded as an epidemic. Class 4, p. 6.
2. Isolation: All known or suspect cases should be isolated promptly.
3. Concurrent disinfection: Collect and burn dressings; autoclave bed clothing, towels, and linens before laundering.
4. Terminal disinfection: Thorough washing with hexachlorophene detergent of all nonexpendable articles in cubicle or unit; boil basins and instruments and autoclave mattresses.
5. Quarantine: None. Surveillance of all suspect or exposed persons including newborn infants.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Examine attendants for evidence of skin lesions and for asymptomatic carrier state.
8. Specific treatment: Use of appropriate antibiotics as determined by bacteriologic tests of sensitivity.

C. Epidemic measures:**1. Isolate all patients and suspects:**

- a. From newborn nurseries: in area separated from regular nursery.
- b. From obstetrical and surgical wards: in separate room.
- c. From general hospital wards: in separate room.

2. Quarantine all exposed newborn infants in area separated from regular nursery, until they can be sent home. Rooming-in may be feasible.**3. Intensive search for missed cases or carriers or source of infection. All cases known or suspect and all attendants caring for such persons should be cultured to determine the presence of staphylococci in nose and throat or lesions. If phage typing is not available, remove and treat all personnel having active lesions.****4. Exclude attendants, nurses, and medical staff with skin or other lesions.****D. International measures: None.**

STREPTOCOCCAL INFECTION, HEMOLYTIC

Group A hemolytic streptococci cause a wide variety of conditions differentiated clinically according to portal of entry and tissue of localization of the infectious agent, also by presence or absence of a scarlatinal rash. The more important conditions are:

A. Scarlet Fever and Streptococcal Sore Throat (Streptococcal Tonsillitis, Streptococcal Pharyngitis)**B. Erysipelas****C. Puerperal Fever**

Streptococcal infections, other than those just mentioned but caused by the same strains of Group A streptococci, include: cellulitis, lymphadenitis, mastoiditis, osteomyelitis, otitis media, peritonitis, septicemia, and various skin and wound infections. Those characterized by purulent exudates are most likely to spread infection, but others such as septicemia are also important because of frequent association with upper respiratory streptococcal carrier states. In so far as these clinical categories are caused by Group A streptococci they are different manifestations of the same infectious agent, and therefore should be treated together in their epidemiologic relationships. They constitute an epidemiologic entity, and similar principles of control hold generally for the group.

A. SCARLET FEVER AND STREPTOCOCCAL SORE THROAT

- 1. Identification**—Scarlet fever is ordinarily streptococcal sore throat in which the infectious agent is capable of producing erythrogenic toxin and the patient has relatively no antitoxic immunity. If the organism is not a good toxin producer, or if the patient is immune to the toxin, the rash does not occur and streptococcal sore throat results. The distinguishing characteristics are fever, sore throat, exudative tonsillitis or pharyngitis, tender cervical adenopathy, leucocytosis, enanthem, strawberry tongue and rash (exanthem). Injection and edema of the pharynx involve the faucial pillars and soft palate, often extending to the hard palate; petechiae are sometimes seen against the background of diffuse redness. Tonsils, if present, often show the exudate of acute follicular tonsillitis. The rash is usually a fine erythema, commonly punctate, blanching on pressure and appearing most often on the neck, chest, in the folds of the axilla, elbow, and groin and on the inner aspects of the thighs.

Typically the rash does not involve the face except in Negroes, but there is flushing of cheeks and circumoral pallor. High fever, nausea, and vomiting accompany severe infections. The desquamation of convalescence is seen at the tips of the fingers and toes and less often over wide areas of the trunk and limbs, including palms and soles. Scarlet fever and streptococcal sore throat may be accompanied or followed by suppurative complications such as otitis media and peritonsillar abscess, and may be followed at an interval of 1 to 4 weeks by nonsuppurative complications such as rheumatic fever and glomerulonephritis. Scarlet fever occasionally occurs in patients with other types of streptococcal infections, such as infected wounds. Severity of the disease has been decreasing in the United States for unknown reasons; fatality is low, about 1 death for each 300 to 400 reported cases. The fatality in some parts of the world is 3 to 5%.

Streptococcal sore throat is scarlet fever infection without a rash. The manifestations of this clinical entity are similar to scarlet fever, except that toxic manifestations including rash do not occur, nor does desquamation follow.

Laboratory diagnostic aids include type demonstration of a Group A streptococcus as the predominant organism in cultures of the throat, and a rise in serum antibody titer (antistreptolysin O, antistreptokinase) from acute to convalescent phase of the illness.

2. **Occurrence**—The clinical disease is common in temperate zones, less so in semitropical areas and rare in tropical climates. Inapparent infections are as common or more common in the tropics than in temperate zones.

In the United States, epidemiological behavior may be endemic, epidemic, or sporadic. Epidemic occurrence is more frequent in certain geographic areas, such as New England, the Great Lakes region, and the Rocky Mountain area. Apart from food-borne epidemics, which may occur in any season, the highest incidence is during late winter and spring. The 5- to 9-year age group is most affected; no sex or racial differences in susceptibility have been defined. Scarlet fever has followed a similar pattern in central Europe, the Scandinavian countries, and Spain during the past 2 decades.

3. **Infectious agent**—*Streptococcus pyogenes*, Group A streptococci, of at least 40 serologically distinct types which vary greatly in geographic and time distributions. Two immunologically different types (A and B) of erythrogenic toxin have been demonstrated.

4. **Reservoir and source of infection**—Reservoir is man; acutely ill or convalescent patients or carriers. Sources of infection are discharges from nose, throat, or purulent lesions, or objects contaminated with such discharges. Nasal carriers are particularly liable to contaminate their environment.

5. **Mode of transmission**—Transmission is by direct contact with patient or carrier, or by indirect contact through objects handled, or by droplet spread whereby streptococci are inhaled; casual contact rarely leads to infection. Streptococci reach the air via contaminated floor dust, lint from bedclothing, personal clothing, handkerchiefs, or occasionally in droplet nuclei discharged by coughing or sneezing; the importance of air-borne transmission and contamination of the environment in spread of infection has not been clearly established. Explosive outbreaks may follow the ingestion of contaminated milk or other food.

6. **Incubation period**—Short, usually 2 to 5 days.

7. **Period of communicability**—In uncomplicated cases, during incubation and clinical illness, approximately 10 days. Thereafter in untreated pa-

tients, communicability decreases progressively, becoming negligible in 2 to 3 weeks although a carrier state may persist for months. Persons with untreated complications resulting in purulent discharges may spread infection for weeks or months. Adequate treatment with penicillin will eliminate probability of transmission from patients or carriers within 24 hours.

- 8. Susceptibility and resistance**—Susceptibility is general, although many persons develop either antitoxic or type-specific antibacterial immunity, or both, through inapparent infection. Antibacterial immunity develops only against the type of Group A streptococcus which induces the patient's disease or inapparent infection, and lasts at least several years. Second attacks of streptococcal sore throat, due to a different type of streptococcus, are not uncommon. The frequency of inapparent infection is unknown, but undoubtedly is related to the prevalence of streptococci, and possibly to the type of streptococcus.

Immunity against erythrogenic toxin, and hence to rash, develops within a week of the onset of scarlet fever and is usually permanent. Second attacks of scarlet fever are rare but may occur because of the 2 immunological forms of toxin.

Both active and passive immunization against erythrogenic toxin are possible but not practical. Neither active nor passive immunization against the streptococcus itself can be accomplished satisfactorily at the present time.

9. Methods of control—

A. Preventive measures:

1. Provision of laboratory facilities for isolation of hemolytic streptococci and the identification of serologic group and type.
2. Emphasis that absence of rash does not decrease the danger of streptococcal infection.
3. Boiling or pasteurization of milk.
4. Exclusion of infected persons from handling milk or other food likely to be contaminated.
5. Milk from any cow with evidence of mastitis should be excluded from sale or use.
6. Chemoprophylaxis with oral sulfonamide drugs (0.5 to 1.0 gram per day) or penicillin (200,000 to 250,000 units twice a day on an empty stomach) for persons to whom recurrent streptococcal infection constitutes a special risk, such as individuals who have had rheumatic fever or chorea within 5 years or are under 18 years of age. See Rheumatic Fever (p. 150). When streptococcal disease occurs in a household which includes a patient with rheumatic fever or person with history of the disease, all members of the family should receive a therapeutic course of penicillin.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report of scarlet fever is required in most states and countries, Class 2A (p. 5). Notification of streptococcal sore throat is inaccurate and has limited value in control because of difficulty in diagnosis and differentiation from nonbacterial exudative tonsillitis and pharyngitis; recommended that reporting be limited to epidemics, Class 4 (p. 6).
2. Isolation: In order of preference, in a single room, cubicle, or small ward; in uncomplicated cases until clinical recovery or not less than 7 days from onset. Isolation may be terminated after 24

hours treatment with penicillin, provided therapy is continued for 7 to 10 days.

3. Concurrent disinfection: Of purulent discharges and all articles soiled therewith.
4. Terminal disinfection: Thorough cleaning; sunning, or other treatment of blankets.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Not indicated in sporadic cases.
8. Specific treatment: Penicillin is the drug of choice. The following regimens have proved effective: (1) benzathine penicillin G, 1 intramuscular injection of 600,000 units (children) or 900,000 units (adults); (2) procaine penicillin G in aluminum monostearate in oil, intramuscular injection of 300,000 units (children) or 600,000 units (adults) every third day for 3 doses; (3) procaine penicillin G as an aqueous suspension, intramuscular injection of 300,000 units daily for 10 days; and (4) penicillin, in the form of a buffered soluble salt of penicillin G, potassium penicillin O, or phenoxymethyl penicillin in doses of 250,000 to 500,000 units given orally 4 times a day for 10 days either 30 minutes before meals or 2 hours after meals. Therapy should be started as early as possible. Such treatment will ameliorate the acute illness, reduce the frequency of suppurative complications, and prevent the development of rheumatic fever and acute glomerulonephritis. The tetracycline antibiotics may be employed in doses of 1 to 2 grams per day for patients sensitive to penicillin, but therapy must be continued for 10 days. Sulfonamide drugs are not as effective as penicillin.

C. Epidemic measures:

1. Determine source and manner of spread, as person-to-person, by milk, or food-borne. Outbreaks can often be traced to an individual or animal with a persistent streptococcal infection through identification of the serologic type of streptococcus.
2. With limited population groups or under special circumstances, penicillin prophylaxis may be given to intimate and household contacts, to those known to have been exposed to contaminated milk or other food, or to the entire population group (see 9A6). In the latter circumstance, sulfonamide drugs should not be administered for prolonged periods because of likelihood that resistant strains of streptococci will develop.
3. Prompt investigation of any group of cases as to possibility of contaminated milk with exclusion of suspected milk supply from sale or use until pasteurized. Contamination of milk or food can occasionally or under special circumstances be determined by bacteriological methods.
4. Stockpiling of penicillin and at least 1 broad-spectrum antibiotic is recommended for use in the event of a disaster situation.

D. International measures: None.

B. ERYSIPELAS

1. Identification—An acute infection characterized by fever, constitutional symptoms, leucocytosis, and a red, tender edematous spreading lesion of the skin, often with a definite raised border. The central point of origin tends to clear as the periphery extends. Face and legs are common sites.

Recurrences are frequent. The disease may be especially severe, with bacteremia, in patients suffering from debilitating disease. Fatality varies greatly with part of body affected and according to associated disease. Rates of 1 to 2% are now common in hospital practice.

Group A streptococci may be isolated from the margin of the skin lesion, the nose and throat, and occasionally from the blood.

Erysipelas due to Group A streptococci is to be distinguished from erysipeloid, caused by *Erysipelothrix rhusiopathiae*, a localized cutaneous infection, primarily an occupational disease of persons handling animals, meat, poultry and fish.

2. **Occurrence**—Geographic and seasonal distributions are similar to scarlet fever and streptococcal sore throat. Common after 20 years of age with highest attack rates at 40 to 60 years, and frequently in infants. No clear predilection for sex or race has been defined. Occurrence is sporadic, even during epidemics of streptococcal infection.
3. **Infectious agent**—*Streptococcus pyogenes*, Group A streptococci, of at least 40 types. No specific strain or type has been shown to cause erysipelas.
4. **Reservoir and source of infection**—Reservoir is man; the source of infection is either respiratory discharges of the same individual or of exogenous origin.
5. **Mode of transmission**—Transmission is by direct contact with patient or carrier, by indirect contact through objects handled, or by droplet spread whereby streptococci are inhaled. Streptococci reach the air through contaminated floor dust, lint from bedclothing, personal clothing, handkerchiefs, or occasionally in droplet nuclei discharged by coughing or sneezing. The importance of air-borne transmission has not been established. Erysipelas may be associated epidemiologically with other forms of Group A streptococcal infection.
6. **Incubation period**—Unknown; probably not more than 2 days.
7. **Period of communicability**—Unknown; presumably until clinical recovery, about 10 days in untreated patients. Adequate treatment with penicillin will eliminate probability of transmission from patients or carriers within 24 hours.
8. **Susceptibility and resistance**—Susceptibility is greatest in infants, in older persons, and in the debilitated. One attack appears to predispose to subsequent attacks; recurrences may be due to streptococcal infection or to hypersensitivity. Whether or not type-specific immunity occurs is unknown. No available immunization procedures.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Personal cleanliness and avoidance of transferring the infectious agent to the broken skin.
 2. Chemoprophylaxis with oral sulfonamide drugs or penicillin as for scarlet fever and streptococcal sore throat.
 3. Recurrence of erysipelas may be prevented by administration of oral sulfonamide drugs or penicillin during and after convalescence, but may need to be continued for months or years depending on the individual situation.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6).
 2. Isolation: During period of communicability; patients are a potential

danger to young infants and to surgical and obstetrical patients. Isolation may be terminated after 24 hours treatment with penicillin, provided therapy is continued for 10 days.

3. Concurrent disinfection: Of dressings and discharges from lesions.
4. Terminal disinfection: Thorough cleaning; sunning or other treatment of blankets.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: None.
8. Specific treatment: Penicillin; procaine penicillin is preferred but oral penicillin G has been used; continue for 7 to 10 days. The tetracycline antibiotics or sulfonamide drugs may be substituted if patient is sensitive to penicillin.

C. Epidemic measures: Erysipelas is now rarely epidemic in the Western world. In the event of an outbreak in a limited population group, institution or hospital, prophylactic administration of penicillin or sulfonamide drugs, as outlined under scarlet fever and streptococcal sore throat.

D. International measures: None.

C. STREPTOCOCCAL PUERPERAL FEVER

1. **Identification**—An acute infection, usually febrile, accompanied by local and general symptoms and signs of bacterial invasion of the genital tract and sometimes of the blood of the postpartum or postabortion patient. Fatality for streptococcal puerperal fever is negligible when adequately treated. **Synonym:** Puerperal Septicemia.

The causative agent can be recovered by bacterial culture of vaginal discharges, cervix and blood and identified by bacteriologic and serologic methods.

A goodly proportion of puerperal infections are of other origin than hemolytic streptococci. They are to be differentiated by appropriate bacteriological means, for they are clinically similar. The infecting microorganisms include a variety of bacterial agents: nonhemolytic streptococci, anaerobic streptococci, *Staphylococcus aureus*, *Escherichia coli*, *Clostridium perfringens*, *Bacteroides* sp., and others. Group A streptococci are of primary importance in postpartum infection; the anaerobic organisms, colon bacilli, *Bacteroides*, sp., and staphylococci in postabortion infections. Treatment of infections caused by bacteria other than hemolytic streptococci is by appropriate antibiotics; the fatality is somewhat greater. Epidemiologic characteristics and methods of control now described for Group A streptococcal infections apply equally to the others.

2. **Occurrence**—World-wide in distribution but reliable morbidity data do not exist. In the United States, mortality has declined more than 80% in the past 15 years, and fatality has dropped precipitously since the advent of antibiotic drugs; greater for white than nonwhite races. Similar changes have occurred throughout the Western world; now chiefly a sporadic disease, although epidemics may occur in institutions where aseptic technics are faulty.
3. **Infectious agent**—*Streptococcus pyogenes*, Group A hemolytic streptococci; hemolytic streptococci of Groups B, C, D, and G. Mixed infections with other bacteria are common.
4. **Reservoir and source of infection**—Reservoir is man; the source of infection is from an external source or from the respiratory tract, in-

testinal tract, genital tract, or skin of the patient. In postpartum infections due to Group A streptococci, the organism comes from an attendant in about half of the cases, from a familial contact in about one-fifth, and from the patient's respiratory tract in the remainder. In postabortum infections, the organism most frequently comes from the patient herself due to deficiencies in aseptic technic.

5. **Mode of transmission**—Direct transfer of infectious agent to uterus may be accomplished by hands or by instruments used in examinations before, during, or following parturition or abortion; transfer of organisms to the genital tract from nose and throat of a carrier or infected attendant, or from the patient's respiratory tract, intestinal tract or skin, is usually by the hands or indirectly as for other streptococcal and wound infection.
6. **Incubation period**—One to three days, rarely longer.
7. **Period of communicability**—During persistence of infective discharges from genital tract of the patient. Infectiousness of the patient herself, in the case of Group A streptococcal infection, will become negligible after 24 to 48 hours of adequate penicillin therapy. In institutional outbreaks contamination of the environment may be extensive and persist for days or weeks.
8. **Susceptibility and resistance**—Susceptibility is general. The birth process and the abortion procedure increase the opportunity for implantation of pathogenic bacteria.
9. **Methods of control**—
 - A. *Preventive measures:*
 1. Maintenance of high standards of prenatal care and extension of such services to all segments of the population.
 2. Strict asepsis in obstetrical procedures with special attention to possible contamination from mouth and nose of attendants as well as by hands and instruments.
 3. Protection of patient during labor and postpartum from attendants, visitors and other patients with respiratory or skin infection.
 4. Bacteriologic search for carriers among attendants, physicians, nurses, and nursemaids.
 5. Prophylactic use of antibiotic drugs in patients undergoing difficult deliveries or sustaining complications that predispose to infection, such as premature rupture of membranes, severe lacerations or retained products of conception.
 6. Education of women in the hazards of self-interruption of pregnancy.
 - B. *Control of patient, contacts, and the immediate environment:*
 1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6).
 2. Isolation: Strict isolation while infectious discharges persist. In patients with Group A streptococcal infection, isolation may be terminated after 24 hours' treatment with penicillin, provided therapy is continued for 7 to 10 days.
 3. Concurrent disinfection: Of dressings and discharges.
 4. Terminal disinfection: Thorough cleaning; sunning or other treatment of blankets.
 5. Quarantine: None.
 6. Immunization of contacts: None.

7. Investigation of contacts and source of infection: Serologic typing of strains of Group A streptococci is of value in tracing the source of infection.
 8. Specific treatment: Penicillin as for other streptococcal infections (see Scarlet Fever 9B8, p. 180). Strict aseptic technic must be used in examination of patients, obtaining of cultures, or surgical interference, since all vaginal manipulations increase the danger of reinfection or of introducing new organisms. In mixed bacterial infections following abortion, one of the broad spectrum antibiotics may be required in addition to penicillin, depending on the infectious agent and sensitivity to the drugs.
- C. Epidemic measures:** The most probable cause of an epidemic of puerperal infection is a Group A streptococcus and the most likely location is a hospital or maternity home. General asepsis should be strictly enforced. The identity and source of the infecting strain of streptococcus should be determined. Infected attendants or carriers should be treated with penicillin. Other personnel and patients may be given oral penicillin prophylactically, 200,000 to 250,000 units twice a day on an empty stomach and continued until the epidemic has subsided.
- D. International measures:** None.

STRONGYLOIDIASIS

1. **Identification**—An infection with a nematode usually involving the mucosa of the duodenum and upper jejunum. Clinical manifestations include dermatitis when the larvae are penetrating the skin, usually of the feet; cough and rales, or even pneumonitis when they are passing through the lungs; abdominal symptoms when the adult females are in the mucosa of the intestine. Symptoms may be mild or severe, depending upon the intensity of the infection. Abdominal symptoms in order of frequency are pain, usually epigastric and often suggesting peptic ulcer, nausea, weight loss, vomiting, diarrhea, weakness, and constipation. Urticaria may occur, especially with reinfection. In rare cases internal autoinfection with hyperparasitism may lead to wasting and death. Eosinophilia is usually moderate, may be absent or intense. Diagnosis is by identifying motile larvae in freshly passed feces, or eggs and motile larvae in fluids obtained by duodenal intubation. Held at room temperature for 24 hours or longer, feces may show developing stages of the parasite, including filariform or infective larvae and nonparasitic male and female adults.
2. **Occurrence**—Geographic distribution closely parallels that of hookworm disease (see *Ancylostomiasis*, p. 23), but apparently indigenous cases occur far beyond the usual confines of that disease, e.g., in Boston, Mass., and Winnipeg, Manitoba. Prevalence in any endemic area is not accurately known.
3. **Infectious agent**—*Strongyloides stercoralis*.
4. **Reservoir and source of infection**—Reservoir is an infected person or dog. Source of infection is moist soil contaminated with feces containing infective (filariform) larvae.
5. **Mode of transmission**—Infective or filariform larvae penetrate the skin, usually of the foot, and are carried to the lungs, where further

development takes place. They penetrate the capillary walls, enter the alveoli, ascend the trachea to the epiglottis and descend the digestive tract to reach the upper part of the small intestine where development of the adult female is completed, the female being generally held to be parthenogenetic. They live embedded in the mucosa of the intestine where eggs are deposited and promptly hatch, liberating rhabditiform larvae which migrate into the lumen of the intestine, leave the host with feces, and develop either into infective filariform larvae or free-living adults. Fertilized females produce eggs which soon hatch, liberating rhabditiform larvae, which become filariform larvae.

6. Incubation period—From penetration of skin by filariform larvae until rhabditiform larvae appear in feces is about 17 days.

7. Period of communicability—As long as living worms remain in the intestine, sometimes for decades.

8. Susceptibility and resistance—Susceptibility to infection is universal. Acquired immunity to strongyloidiasis has been demonstrated in laboratory animals but not in man.

9. Methods of control—

A. Preventive measures:

1. As for Ancylostomiasis (p. 24). The installation and strict use of sanitary disposal systems for human excreta, particularly sanitary privies in rural areas, are fundamental preventive measures.
2. Rigid attention to hygienic habits, particularly the wearing of shoes.
3. Treat dogs with cyanine dyes. Destroy stray dogs.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in most states and countries not a reportable disease, Class 3C (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: Sanitary disposal of excreta.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Of family contacts for evidence of infection.
8. Specific treatment: Cyanine dyes (vanquin® and dithiazanine).

C. Epidemic measures: Not applicable, a sporadic infection.

D. International measures: None.

SYPHILIS

Two distinctive forms of syphilis are recognized. That form which is venereally spread is of world-wide occurrence. The other, often referred to as endemic syphilis or bejel is of nonvenereal spread and confined to parts of the world where economic, social, and climatic conditions favor its development; not seen in the United States.

A. VENEREAL SYPHILIS

1. Identification—An acute and chronic relapsing treponematoses characterized clinically by a primary lesion, a secondary eruption involving skin and mucous membranes, long periods of latency, and late lesions of

skin, bone, viscera, the central nervous, and cardiovascular systems. The primary lesion appears at about 3 weeks as a papule and after erosion presents a variety of forms, the most distinctive although not the most frequent being an indurated chancre; invasion of the blood precedes the initial lesion; a hard nonfluctuant painless satellite bubo commonly follows. Infection without chancre is fairly frequent. During the next 4 to 6 weeks, even without specific treatment the chancre begins to involute and the generalized secondary eruption appears, often accompanied by mild constitutional symptoms. Secondary manifestations disappear spontaneously within a few weeks to as long as 12 months, with subsequent clinical latency of weeks to years, often interrupted in early years by recurrence of infectious lesions of skin and mucous membrane or developing lesions of the eye and central nervous system; in later years (5 to 20) by explosive destructive noninfectious lesions of skin, bone, and mucosal surfaces. Latency sometimes continues through life, sometimes spontaneous recovery occurs, and in other instances and unpredictably late disabling manifestations in cardiovascular, central nervous or other systems appear. The actual fatality is unknown; prenatal infection is frequently fatal, before birth or in infancy. Early acquired syphilis does not result in death or serious disability but the late manifestations shorten life, impair health, and limit occupational efficiency.

Primary and secondary syphilis are confirmed by dark-field examination of exudates of lesions, and in all instances by serologic tests for syphilis through examination of blood and spinal fluid. These tests may be supplemented by treponemal immobilization or agglutination tests to aid in the exclusion of biologic false-positive reactions. The dark-field examination is indispensable in seronegative primary syphilis.

2. **Occurrence**—One of the more frequent communicable diseases; widespread throughout the world and primarily involving young persons between ages 15 and 30 years. The considerable differences in racial incidence are related more to social than to biologic factors. More prevalent in urban than rural areas and in males more than females.
3. **Infectious agent**—*Treponema pallidum*.
4. **Reservoir and source of infection**—Man is the only reservoir. Source of infection is exudates from obvious or concealed moist early lesions of skin and mucous membrane of infected persons; body fluids and secretions (saliva, semen, blood, vaginal discharges) during infectious period.
5. **Mode of transmission**—By direct contact (sexual intercourse, kissing, fondling of children) during primary and secondary syphilis. Transmission through indirect contact with contaminated articles has relatively little significance. Prenatal infection may occur after the fourth month of pregnancy through placental transfer of treponemata; transmission occasionally occurs through blood transfusion.
6. **Incubation period**—Ten days to 10 weeks, usually 3 weeks.
7. **Period of communicability**—Variable and not definitely known; during primary and secondary stages, also during mucocutaneous recurrence intermittently during 2 to 4 years. Extent of communicability through sexual intercourse during latent period (2 to 4 years) has not been established; possible inapparent lesions require that this stage be considered potentially infectious. Adequate treatment usually ends infectivity within 24 hours.
8. **Susceptibility and resistance**—Susceptibility is universal; no natural immunity. Infection leads to gradually developing resistance against the homologous strain of treponema and to an extent against heterologous strains; may be overcome by large reinfecting doses or fail to de-

velop by reason of treatment in primary or secondary stages. Superinfection may produce lesions simulating those of the currently existing stage; in late latency, superinfection has special significance through ability to produce benign late lesions of skin and mucous membrane.

9. Methods of control—

A. Preventive measures: The following are applicable to all venereal diseases: syphilis, chancroid, lymphogranuloma venereum, granuloma inguinale, and gonorrhea.

1. General health promotional measures, health and sex education, preparation for marriage, premarital and prenatal examinations as part of general physical examination. Improvement of social and economic conditions, including recreational facilities.
2. Protection of the community by suppression of commercialized prostitution and of clandestine sexual promiscuity, in cooperation with social and law enforcement agencies; by teaching methods of personal prophylaxis applicable before, during, and after exposure; by repeated prenatal serologic examination of all pregnant women.
3. Provision of facilities for early diagnosis and treatment; encouragement of their use through education of the public concerning symptoms of the venereal diseases and modes of spread, and through making these services available irrespective of economic status of the infected person. Intensive case-finding programs, to include interview of patients and tracing of contacts, and repeated mass serologic examination of special groups with known high incidence of venereal disease.
4. An emphasis on control of patients with venereal disease in a transmissible stage should not preclude search for persons past that stage; has value in preventing relapse, congenital syphilis and disability due to late manifestations.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report is required in all states of the United States and variously in other countries, Class 2B (p. 5). Reporting in the United States is commonly too incomplete to be a reliable guide to administrative practice. The need for accurate reporting increases as syphilis declines, in order to determine the amount of effort to be expended on control activities.
2. Isolation: None. To avoid reinfection, patients should refrain from sexual intercourse with previous partners not under treatment.
3. Concurrent disinfection: None in adequately treated cases; care in disposal of discharges from open lesions and articles soiled therewith.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Not applicable.
7. Investigation of contacts and source of infection: Interview of patients and tracing of contacts are the fundamental features of a program for control of venereal disease. Trained interviewers obtain the best results. The stage of the disease determines criteria for contact tracing: (a) for primary syphilis, all sexual contacts of preceding 3 months; (b) for secondary syphilis, those of the preceding 6 months; (c) for early latent syphilis, those of the preceding year provided time of primary and secondary lesions is not established; (d) for late and late latent syphilis, marital partners and children of infected mothers; (e) for congenital syphilis, all members of immediate family.

8. Specific treatment: Long-acting penicillin (PAM, benzathine penicillin or equivalent preparations); in general, large amounts should be given initially on the day of diagnosis to assure reasonably effective therapy should the patient fail to return. Penicillin sensitive persons may be treated with erythromycin or the tetracycline antibiotics. Such patients require extended post-treatment observation.

C. Epidemic measures: Intensification of measures outlined under 9A and 9B.

D. International measures:

1. Appropriate examination of groups of adolescents and young adults moving from areas of high prevalence of treponemal infections.
2. Adherence to agreements among nations (e.g., Brussels agreements) as to records, and provision of diagnostic, treatment and contact interviewing facilities at seaports for foreign seamen engaged in maritime commerce.
3. Provision for rapid international exchange of information on contacts.

B. NONVENEREAL SYPHILIS

1. **Identification**—A disease of limited geographical occurrence and distribution, acute nature, and characterized clinically by an eruption of skin and mucous membrane, usually without evident initial primary sore. Mucous patches of the mouth are often the first lesions, soon followed by moist papules in folds of the skin and by drier lesions of trunk and extremities. Other early skin lesions are macular or papular lesions, often hypertrophic and frequently circinate, and resembling those of venereal syphilis. Plantar and palmar hyperkeratoses occur frequently, often with painful fissuring; a patchy depigmentation and hyperpigmentation of the skin and alopecia are common. Inflammatory or destructive lesions of skin, long bones, and nasopharynx are late manifestations. Unlike venereal syphilis, the nervous and cardiovascular systems are rarely involved. Case fatality is negligible. Synonyms: Bejel, Dichuchwa, Njovera, Sibbens, Radesyke.

Serologic tests for syphilis are reactive in the early stages and remain so for many years of latency, gradually tending toward reversal; response to treatment as in venereal syphilis.

2. **Occurrence**—A common disease in localized areas, where a low standard of education and poor economic and social conditions prevail, with primitive sanitary and dwelling arrangements. Present in the Balkans, in Turkey, and in eastern Mediterranean countries. Numerous foci in Africa, particularly in arid regions.
3. **Infectious agent**—*Treponema pallidum*.
4. **Reservoir and source of infection**—Reservoir is man. Source of infection is early lesions of skin and mucous membrane of infected persons.
5. **Mode of transmission**—Direct or indirect contact with infectious lesions is favored by common use of eating and drinking utensils and generally unsatisfactory hygienic conditions. Congenital transmission is rare.
6. **Incubation period**—Two weeks to 3 months, usually about 6 weeks.
7. **Period of communicability**—During periods of moist eruptions of skin and until mucous patches disappear; may extend over several weeks or months.
8. **Susceptibility and resistance**—Similar to venereal syphilis.
9. **Methods of control**—The considerable decline in overt early venereal syphilis throughout much of the world calls for particular attention to nonvenereal treponematoses, not yet under control, and including Yaws, p. 221; and Pinta, p. 127.

A. Preventive measures:

1. Those of the nonvenereal treponematoses. See Yaws 9A, p. 221.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in most countries not a reportable disease, Class 3B (pp. 5, 6).
2. See Yaws 9B for items 2–8, applicable to all nonvenereal treponematoses.

C. Epidemic measures: Intensification of preventive and control activities.**D. International measures:** See Yaws 9D.

TAENIASIS AND CYSTICERCOSIS

1. **Identification**—*Taenia* infection of man is manifest in two forms, the one a benign intestinal infection with the adult worm of one or other of two species, beef and pork tapeworms, and the other a severe somatic disease (cysticercosis) of many different tissues arising through localization of larvae of the pork tapeworm.

Clinical manifestations of infection with the adult worm are variable, frequently vague or absent; sometimes nervousness, insomnia, anorexia, loss of weight, abdominal pain, and digestive disturbances. A nonfatal disease. Synonym: Beef or Pork Tapeworm Infection.

Infection with adult tapeworms is confirmed through identification of proglottides (segments) of the worm or of eggs in feces. Specific diagnosis is through morphological features of the gravid proglottides (Strobila), often obtained after treatment; obtaining the scolex or head confirms the identification and assures elimination of the worm.

If eggs of the pork tapeworm are swallowed by man, they hatch in the small intestine and larval forms (cysticerci) develop in the subcutaneous tissues, striated muscles and other regions of the body. Grave consequences can ensue when they localize in heart, eye, or central nervous system. Recognition of subcutaneous or somatic cysticercosis is by excision of the larva and microscopic examination. In the presence of cerebral symptoms, somatic cysticercosis strongly suggests cerebral involvement. Roentgen ray examination is helpful in locating calcified cysticerci in the brain and muscle, and in determining the intensity of infection. A chronic disease of relatively high fatality.

Infections with *Hymenolepis nana*, *H. diminuta*, and *Dipylidium caninum* are relatively rare; the eggs must be differentiated from *taenia* eggs.

2. **Occurrence**—Cosmopolitan distribution; particularly frequent wherever beef or pork is eaten raw or slightly cooked. Incidence is highest in East Africa, Tibet, Mexico, Peru, and eastern Europe. Where *Taenia saginata* and *Taenia solium* co-exist, *T. saginata* is by far the commoner. Infection with *T. solium* is rare in the United States and Canada.
3. **Infectious agents**—*Taenia saginata*, the beef tapeworm of man, intestinal infection with adult worm only; *Taenia solium*, the pork tapeworm of man, intestinal infection with adult worm and somatic infection with the larvae, *Cysticercus cellulosae*.
4. **Reservoir and source of infection**—Reservoir is an infected person discharging eggs of the parasite in feces. Immediate source of infection for *T. saginata* is the flesh of infected cattle; for intestinal taeniasis due to *T. solium* the flesh of infected pigs. Source of infection for cysticercosis of man is the eggs in feces of an infected person.

5. **Mode of transmission**—For *Taenia saginata*, by ingestion of raw or inadequately cooked beef containing the infective larva—a cysticercus. For *Taenia solium* (1) by ingestion of raw or inadequately cooked pork containing the infective larva (cysticercus) and subsequent development of the adult worm in the intestine; or (2) by direct hand to mouth transfer of eggs in feces of an infected person or indirectly through ingestion of food or water contaminated with eggs, resulting in somatic cysticercosis.
6. **Incubation period**—From 8 to 10 weeks.
7. **Period of communicability**—The adult worm of *T. saginata* is not transmissible under natural conditions from man to man; eggs are disseminated in the environment as long as man harbors the worm in the intestine. Man disseminates eggs of *T. solium* as long as the worm is harbored in the intestine, sometimes 30 to 40 years. Transmission of adult worms from man to man is impossible.
8. **Susceptibility and resistance**—Man is universally susceptible. No apparent resistance follows infection.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Prevention of soil contamination with human feces in rural areas and education of the public. Proscribe use of sewage effluents for pasture irrigation.
 2. Thorough cooking of beef and pork insures against infection.
 3. Adequate inspection of the carcasses of cattle and swine can detect most infected meat, although in beef cysticerci may be few and escape attention.
 4. Immediate treatment of persons harboring adult *T. solium* is essential to prevent human cysticercosis.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
 2. Isolation: None; patients with *T. solium* infection should be excluded from preparation and serving of food.
 3. Concurrent disinfection: Sanitary disposal of feces; for *T. solium* rigid sanitation with washing of hands after defecating and before eating.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Usually not a profitable procedure.
 8. Specific treatment: Quinacrine hydrochloride and oleoresin of aspidium. No specific therapy for cysticercosis.
 - C. **Epidemic measures:** None.
 - D. **International measures:** None.

TETANUS

1. **Identification**—An acute disease induced by toxin of the tetanus bacillus growing anaerobically at site of an injury; characterized by painful muscular contractions, primarily of masseter and neck muscles, secondarily of trunk; rigidity is sometimes confined to the region of injury.

History of injury and known portal of entry sometimes lacking. Much variation in fatality according to age and length of incubation, average about 35%.

2. **Occurrence**—World-wide, but relatively uncommon. An occasional disease among farmers in central United States, especially following wounds contaminated with manured soil. Is a serious factor in infant mortality where midwives are ignorant or incompetent. Formerly an important disease in military practice, now effectively controlled by active immunization.
3. **Infectious agent**—*Clostridium tetani*, tetanus bacillus.
4. **Reservoir and source of infection**—Reservoir is infected domestic animals, especially horses, also man. The immediate source of infection is soil, street dust, or animal and human feces.
5. **Mode of transmission**—Tetanus spores enter the body through injury, usually a puncture wound, but also burns and trivial or unnoticed wounds. Tetanus neonatorum usually occurs through infection of the unhealed umbilicus.
6. **Incubation period**—Commonly 4 days to 3 weeks, dependent somewhat upon character, extent and location of wound; longer periods have been noted.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man.
8. **Susceptibility and resistance**—Susceptibility is general. Active immunity is induced by tetanus toxoid, passive immunity by tetanus antitoxin.

9. Methods of control—

A. Preventive measures:

1. Community education about the danger of certain types of injury, the value of routine immunization, and the need after injury for either a reinforcing (booster) injection if previously actively immunized, or passive protection by tetanus antitoxin if not immunized.
2. Active immunization with tetanus toxoid gives solid and satisfactory protection against tetanus. It has the advantage of protection against injuries erroneously considered as not warranting protective measures. The initial inoculation is preferably in infancy or early childhood, given in combination with diphtheria toxoid and pertussis vaccine. Tetanus toxoid is also recommended for workers in contact with soil or domestic animals and for military forces, policemen, firemen, and others specially liable to traumatic injury. Pregnant women should be actively immunized in regions where tetanus neonatorum is prevalent.

Number of doses and intervals between injections in the initial series are according to recommendations in Diphtheria 9A1, p. 67, these being determined by the particular form of toxoid used. A reinforcing (booster) injection should be given about 8 to 12 months later; and thereafter, in the absence of injury, at intervals no longer than 5 years. An inoculated person desirably should have with him at all times a record of tetanus immunization.

3. If a person previously actively immunized against tetanus has an injury with danger of tetanus, especially a puncture wound contaminated with dirt, a single reinforcing (booster) injection of tetanus toxoid is administered promptly on the day of injury; reactions are essentially absent. Such procedure has great advantage over passive immunization with tetanus antitoxin. It obviates risk

of horse serum reactions, of special importance to persons known to be allergic to a variety of substances.

4. In the absence of previous active immunization, passive protection of injured persons is indicated through injection of 3,000 to 5,000 units of tetanus antitoxin, providing the patient is seen on the day of injury and there are no compound fractures, gunshot wounds or other wounds not readily debrided. If the delay is greater or such complications are present, the recommended dose is 6,000 to 10,000 units according to the particular circumstance. Half the stated dosage suffices for children under 10 years of age. The protection lasts about 10 days. Because a reaction to horse serum is far more likely should the emergency again arise, active immunization should follow passive protection; concurrently if the dose of antitoxin was no more than 3,000 units, after several weeks with larger amounts.
 5. Under all circumstances, foreign matter is to be removed from wounds by thorough cleaning, with debridement where indicated.
 6. Licensing of midwives, with professional supervision and education as to methods, equipment, and technic of asepsis.
- B. Control of patient, contacts, and the immediate environment:**
1. Report to local health authority: Case report required in most states and countries, Class 2A (p. 5).
 2. Isolation: None.
 3. Quarantine: None.
 4. Immunization of contacts: None.
 5. Investigation of contacts and source of infection: Case investigation to determine circumstances of injury.
 6. Concurrent disinfection: None.
 7. Terminal disinfection: None.
 8. Specific treatment: Tetanus antitoxin in a single large dose intravenously; penicillin in large doses intramuscularly. Sedation is important.
- C. Epidemic measures:** Thorough search for inadequacies in technic of sterilization in the uncommon hospital outbreaks; in tetanus neonatorum, rigid inquiry into competence and licensure of attendants at birth.
- D. International measures:** Active immunization against tetanus is advised for international travelers.

TOXOPLASMOSIS

1. **Identification**—A protozoan infection acquired prenatally from the mother or at any time postnatally. Early prenatal infection may lead to death of the fetus or to residual manifestations at birth or subsequently of chorioretinitis, cerebral calcification, hydrocephalus, microcephalus, psychomotor retardation or convulsions. Prenatal infection still active at birth may be manifested postnatally by fever, jaundice, rash, hepatomegaly, splenomegaly, xanthochromic spinal fluid and convulsions. Infections acquired after birth may be so mild as to be unrecognized; or slightly more severe with fever, lymphadenopathy and lymphocytosis

persisting for days or weeks, less often in severe form with generalized muscle involvement, cerebral manifestations, and death. A brief maculopapular eruption may accompany the acquired disease and is unrelated to clinical severity of the infection.

Definitive diagnosis is by microscopic demonstration of *Toxoplasma* in body tissues or fluids during life or at autopsy; or in laboratory reared albino mice inoculated with such materials. Other laboratory aids are complement-fixation tests, methylene blue dye test, hemagglutination test and fluorescence inhibition test.

2. **Occurrence**—Distribution world-wide in animals and man. Infection in man is apparently common but clinical disease is exceptional.
3. **Infectious agent**—*Toxoplasma gondii*, a protozoan parasite.
4. **Reservoir and source of infection**—Rodents, dogs, cats, swine, cattle, sheep, goats, chickens, and other mammals and birds are reservoirs; a most ubiquitous parasite. The source of human infection is unknown.
5. **Mode of transmission**—Unknown with regard to postnatal infections. Congenital infection is apparently through the placenta of pregnant women who are in the primary stage of the infection. Infection also possible during delivery. No arthropod vector has been established although ticks infected in the laboratory have transmitted infection to experimental animals.
6. **Incubation period**—Unknown; probably from about 2 weeks to several months.
7. **Period of communicability**—Under natural conditions probably not directly transmissible from man to man except during gestation. In animals, probably during acute stage and possibly longer.
8. **Susceptibility and resistance**—Susceptibility is general. Recovery from one attack probably confers permanent immunity.
9. **Methods of control**—

A. Preventive measures:

1. Suggestive preventive measures are to avoid intimate contact with sick animals and birds, avoid tick bites, and keep premises free from rats and mice. No specific preventive measures are known.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: In congenital cases, determine antibodies in mother and other members of family; in acquired cases, determine contact with infected animals and history of bite by arthropods.
8. Specific treatment: In experimental infections of animals, sulfonamides have prophylactic value, and when administered early may have a curative effect. A combination of pyrimethamine (daraprim®) with triple sulfonamides has given better results. The results in man are discouraging.

C. Epidemic measures: Not applicable, a sporadic disease. Community surveys and voluntary reporting by hospitals, especially ophthalmological

clinics, are needed to estimate incidence, and along with case study, to obtain information leading to better knowledge of the disease.

D. International measures: None.

TRACHOMA

1. **Identification**—A chronic communicable disease of the eye characterized by abrupt or insidious onset with inflammation of the conjunctivae and subepithelial infiltration, followed by follicular hypertrophy of the conjunctiva, capillary infiltration of the cornea (pannus) and by cicatrization leading to gross deformity of the eyelids, visual disability, and possibly blindness. Secondary bacterial infections contribute to variations in clinical and epidemiological behavior of the disease.
Laboratory diagnosis is by finding cytoplasmic inclusion bodies, and by cytological changes in expressed follicular material.
A number of forms of chronic follicular conjunctivitis of bacterial origin simulate trachoma and require differentiation, also epidemic keratoconjunctivitis, p. 97.
2. **Occurrence**—World-wide, in tropical, subtropical, temperate, and cold zones, but with unequal and varying distributions, marked by differences in age of onset, clinical evolution, frequency of spontaneous cure, frequency of disabling sequelae, and response to treatment. Trachoma is widespread in the Middle East, in Asia, and along the Mediterranean littoral; formerly was common from West Virginia to Oklahoma in the United States but presently occurs only as sporadic cases in this area and among Indians of the Southwest. High prevalence is generally associated with poor hygiene, poor nutrition, poverty, and crowded living conditions, particularly in dry, dusty areas. Nomadic persons seem in general to suffer a less severe form of trachoma than stationary populations.
3. **Infectious agent**—The virus of trachoma, one of the psittacosis-lymphogranuloma group.
4. **Reservoir and source of infection**—Reservoir is man; source of infection is secretions from the eyes, and mucoid or purulent discharges of nasal mucous membranes of infected persons; tears of such persons also carry infection.
5. **Mode of transmission**—By direct contact with secretions of infected persons or by indirect contact with materials recently soiled therewith. Flies may contribute to spread of the disease in Middle East and Far East countries, and rather clearly to dissemination of the associated conjunctivitis. Carriers have not been demonstrated. Communicability is relatively low.
6. **Incubation period**—From 5 to 12 days as determined by human volunteer experiments.
7. **Period of communicability**—While active lesions are present in the conjunctivae and in the annexed mucous membranes. Communicability no longer exists after complete cicatrization, but reactivation of the disease may occur with reappearance of infective discharges.
8. **Susceptibility and resistance**—Susceptibility is general; in all races affects children more frequently than adults, particularly persons of unclean habits and those whose eyes are irritated by exposure to sun, wind, and sand. Natural or acquired immunity has not been demonstrated.

9. Methods of control—

A. Preventive measures:

1. In areas where trachoma is prevalent, careful and systematic examination of the eyes of children, especially pre-school children.
2. The use in common of toilet articles and towels should be prohibited.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report required in most states and countries, Class 2B (p. 5).
2. Isolation: Children with active lesions should be excluded from school when adequate preventive measures are not practicable. With proper instruction of patient and family in means of preventing spread, and with adequate treatment of the patient, no need exists for isolation.
3. Concurrent disinfection: Of eye discharges and articles soiled therewith.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Members of the family, playmates, and schoolmates.
8. Specific treatment: Tetracycline antibiotics in oil or ointment applied locally are effective in most instances. Oral sulfonamides may be used for resistant cases.

C. Epidemic measures: In regions of hyperendemic prevalence mass treatment campaigns have been successful in reducing frequency of the disease, when associated with health education of the people and effort toward improvement of sanitary conditions of the environment.

D. International measures: None.

TRICHINOSIS

1. Identification—An infection arising through invasion of human and animal hosts by larvae (trichinae) of a parasitic nematode, *Trichinella spiralis*. Clinical disease in man is markedly irregular, severity varying with number of trichinae, tissue invaded and physiological state of the host. Sudden appearance of edema of upper eyelids is a common, early, and characteristic sign of clinical trichinosis, usually about the eleventh day of infection, and sometimes followed by subconjunctival and retinal hemorrhage, pain, and photophobia. Gastrointestinal symptoms may precede or accompany ocular manifestations. Muscle soreness and pain, skin lesions, thirst, profuse sweating, chills, weakness, prostration and rapidly increasing eosinophilia may shortly follow ocular signs. Fever is usual, remittent, and terminates by lysis after about a week; sometimes as much as 40° C (104° F) for several days. Respiratory and neurological symptoms may appear in the third to sixth week. Myocardial failure, when it occurs, is between the fourth and eighth weeks. Trichinosis is usually a mild febrile disease. Synonyms: Trichiniasis, Trichinellosis.

Daily study of blood smears for increasing eosinophilia is the most useful diagnostic laboratory procedure. Skin tests, flocculation tests,

and complement-fixation tests may aid diagnosis but are not in themselves conclusive. Search for parasites in feces, blood, spinal fluid, and biopsied striated muscle is often futile.

2. **Occurrence**—World-wide, but rare or absent in native populations of the tropics or where swine are fed on root vegetables, as in France. The parasite is particularly widespread in the United States, about 1 in every 6 necropsies showing infection; less prevalent in Canada and Mexico. The disease is no longer prevalent in western Europe but is still common in eastern Europe. Outbreaks recently reported in Argentina. Clinical cases occur more frequently than indicated by morbidity reports; often confused with other illnesses and patients with mild infection do not seek medical aid. No selection by age, sex, race, region, season, or climate except as these affect the eating of insufficiently cooked flesh of infected hogs or other animals.
3. **Infectious agent**—*Trichinella spiralis*.
4. **Reservoir and source of infection**—Swine and many wild animals. fox, wolf, bear, polar bear, marine mammals, and rats are reservoirs of infection. The source of infection is meat of infected animals, chiefly pork and pork products, occasionally that of bears and marine mammals.
5. **Mode of transmission**—By eating flesh of animals containing viable trichinae. The cysts develop into sexual worms in the intestines. The female worm then passes larvae which penetrate the intestinal wall and enter the lymphatics. They are carried from there to various muscles and organs where they encyst.
6. **Incubation period**—About 9 days after ingestion of infective meat with variation between 2 and 28 days.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Animal hosts remain infective for many years, and meat from such animals for appreciable periods unless refrigerated at low temperatures (see 9A5).
8. **Susceptibility and resistance**—Susceptibility is universal. Infection is not known to result in acquired immunity, but partial resistance may be induced.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Inauguration of regulations and enforcement to assure adequate processing of pork products. Meat inspection for trichinosis is impractical, costly, and gives a false sense of security.
 2. Elimination of practice of feeding uncooked garbage and offal to swine and the adoption and enforcement of suitable laws and regulations insuring cooking such material before its consumption by swine.
 3. Cooking of all fresh pork and pork products by the consumer at a temperature and for a time sufficient to allow all parts of the meat to reach at least 65.6° C (150° F) (a temperature which allows a good margin of safety) unless established that these meat products have been processed under official regulations assuring adequate destruction of trichinae.
 4. Low temperatures maintained in central portions of pork are believed effective in killing trichina larvae, such as -27° C (-16° F) for 36 hours. Storage in home freezers as a safeguard against trichinosis is not to be depended upon.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report desirably required in most states and countries, Class 2B (p. 5).
2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: More reasonably a community than a case effort.
8. Specific treatment: None.

C. Epidemic measures: Establish the diagnosis. Examine histories and institute epidemiologic study to determine the common food involved. Destroy remainder of food and initiate measures to correct faulty practices.**D. International measures:** None.

TRICHOMONIASIS

1. **Identification**—A common non-fatal chronic infection of the genitourinary tract characterized in women by vaginitis associated with profuse leukorrhea and thin, foamy, yellowish discharge of foul odor. The vaginal mucosa is inflamed, frequently with small petechial or punctate hemorrhagic lesions. In men, the infectious agent lives in prostate, urethra, or prepuce, rarely producing symptoms or demonstrable lesions.

Diagnosis is through identification of the motile parasite, by immediate microscopic examination of discharges, or by culture.

2. **Occurrence**—In selected areas of the United States the incidence among Negroes is twice that of whites. Geographically widespread, and a frequent disease of all continents and all peoples, primarily of adults, with highest incidence among young girls and women aged 16 to 35 years.
3. **Infectious agent**—*Trichomonas vaginalis*, a protozoan.
4. **Reservoir and source of infection**—Reservoir is man; vaginal and urethral discharges of infected persons are sources of infection.
5. **Mode of transmission**—By sexual intercourse with infected persons; possibly by contact with contaminated articles.
6. **Incubation period**—Four to 20 days, average 7 days.
7. **Period of communicability**—For the duration of the infection.
8. **Susceptibility and resistance**—General and high grade susceptibility to infection but clinical disease is mainly in females.
9. **Methods of control**—

A. Preventive measures:

1. Avoidance of sexual intercourse with known infected individuals.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).

2. Isolation: None; avoid sexual relations during period of infection and treatment.
3. Concurrent disinfection: None; the organism cannot withstand drying.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Marital partner, particularly if a recurrent infection.
8. Specific treatment: Most infected females respond to a variety of chemotherapeutic agents, including various arsenicals, sulfonamides, broad-spectrum antibiotics and oxyquinoline derivatives, used locally in conjunction with a maintained vaginal acidity and cleanliness.

C. Epidemic measures: None.

D. International measures: None.

TRICHURIASIS

1. **Identification**—A nematode infection of the large intestine. Light infections are often asymptomatic and detected only when fecal examination reveals eggs. With heavy infections, intermittent abdominal discomfort, emaciation, and anemia are observed. Synonyms: Trichocephaliasis, Whipworm Disease.

Diagnosis of infection is by demonstration of eggs in the feces.

2. **Occurrence**—Cosmopolitan, especially in warm, moist regions.
3. **Infectious agent**—*Trichuris trichiura* (*Trichocephalus trichiurus*), the human whipworm.
4. **Reservoir and source of infection**—Reservoir is an infected person discharging eggs in feces. The source of infection is soil contaminated with human feces and embryonated eggs of the parasite.
5. **Mode of transmission**—Indirect, in that eggs passed in feces require at least 10 days outside the body for embryonation in soil. Ingestion of fully embryonated eggs is followed by hatching and attachment of the developing worm to the mucosa of the cecum and proximal colon.
6. **Incubation period**—From ingestion of eggs to passage of eggs, about 90 days; the incubation period, until symptoms appear, is longer, variable and indefinite.
7. **Period of communicability**—As long as eggs are passed in feces, many years.
8. **Susceptibility and resistance**—Susceptibility is universal.
9. **Methods of control**—

A. Preventive measures:

1. Provision of adequate facilities for proper disposal of feces.
2. Education of all members of the family, particularly children, to use toilet facilities.
3. Encouragement of satisfactory hygienic habits on the part of children, especially the practice of washing the hands before handling food.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Official report not ordinarily justifiable. Class 5 (p. 6). School health authorities should be advised of unusual frequency in school populations.

2. Isolation: None.
 3. Concurrent disinfection: None; sanitary disposal of feces.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Fecal examination of members of family group, especially children, and of playmates.
 8. Specific treatment: Cyanine dye (Dithiazanine).
- C. *Epidemic measures*: Not applicable.
- D. *International measures*: None.
-

TRYPANOSOMIASIS, AFRICAN

1. **Identification**—A protozoan disease confined to tropical Africa, characterized in its early stage by fever, intense headache, insomnia, lymph node enlargement (especially posterior cervical), anemia, local edema, and rash; and in its later stages by wasting, somnolence, and other symptoms due to involvement of the central nervous system. The disease may run a protracted course of several years, or death may follow within a few months; a highly fatal disease. Synonym: African Sleeping Sickness.
Diagnosis in the early stages is by finding trypanosomes in lymph from punctured lymph nodes; less readily in the peripheral blood. In late stages of sleeping sickness, trypanosomes are present in cerebrospinal fluid. Inoculation of blood or lymph into rats, guinea pigs, or monkeys, or culture on appropriate media are sometimes useful.
2. **Occurrence**—The disease is confined to tropical Africa between 15° N and 20° S latitude, corresponding to distribution of the tsetse fly. A prevalence of 30% has been demonstrated in some regions. Epidemics tend to occur when the disease is introduced into nonimmune populations. In regions where *Glossina palpalis* is the principal vector, infection occurs mainly along streams (Gambia, Liberia, Sierra Leone, Ghana, Congo, Sudan, Uganda). Where *G. morsitans* is the principal vector, infection is over wider dry areas (Mozambique, Nyasaland, Rhodesia, Tanganyika).
3. **Infectious agents**—*Trypanosoma gambiense* and *T. rhodesiense*, hemoflagellates.
4. **Reservoir and source of infection**—Man is the main reservoir of both *T. gambiense* and *T. rhodesiense*, the infectious agent being present in the blood. Wild game, especially bushbuck and antelopes, and domestic cattle and pigs are the chief animal reservoirs. The immediate source of infection is an infective tsetse fly.
5. **Mode of transmission**—By the bite of certain species of *Glossina*, tsetse flies. Four species are mainly concerned, *G. palpalis*, *G. tachinoides*, *G. morsitans*, and *G. swynnertoni*. In nature, the first two transmit *T. gambiense* and the latter two *T. rhodesiense* infections, although in the laboratory many other species transmit both agents. The tsetse fly is infected by biting an infected person or animal. The parasite develops in the fly in 18 days or longer according to temperature and other factors. Infection is conveyed by the bite. Direct mechanical transmission by blood on proboscis of *Glossina* is presumably possible. Once infected, a tsetse fly remains infective for life, up to 3 months; infection

is not passed from generation to generation. A few cases of congenital infection in man have been reported.

6. **Incubation period**—Usually 2 to 3 weeks. May be as short as 7 days.
7. **Period of communicability**—As long as the parasite is present in the blood of the infected person; extremely variable in untreated cases and in late as well as in early stages of the disease.
8. **Susceptibility and resistance**—Susceptibility is general. Rare cases of spontaneous recovery without symptoms of central nervous system involvement have been reported; also some few untreated advanced cases with nervous system disease. Inapparent infection with *T. gambiense* is known.

9. Methods of control—

A. Preventive measures:

1. Mass survey of whole local populations with treatment of all infected persons in order to reduce the human reservoir of infection.
2. Wide and if necessary repeated clearings of bush around villages, along lines of communication, especially streams near houses and roads. More likely to be successful against *G. palpalis* than other species.
3. Concentration of population in relatively large villages is occasionally indicated but only when associated with other large-scale improvements for the health and economy of the community.
4. Tsetse fly control by all practical means, including use of insecticides with residual effect, and trapping.
5. Segregation of big game into game reserves away from human habitation (for *G. morsitans*).
6. Education of the population as to mode of spread of the disease and methods of prevention.
7. Individual protection can be achieved temporarily by chemoprophylaxis. A single adult dose of 250 mg. of pentamidine-isethionate intramuscularly protects for 3 to 6 months.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas to obtain records of prevalence and to encourage control measures; not a reportable disease in most countries, Class 3B (pp. 5, 6).
2. Isolation: Patients with trypanosomes in the blood should be protected from bites of tsetse flies; isolation is not practicable. In some countries legal restrictions are placed on movement of untreated patients.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Should be done in connection with study of infected population groups. Not profitable for the individual case.
8. Specific treatment: *T. rhodesiense* infection is much more resistant to chemotherapy than *T. gambiense*. Before the nervous system is involved, Bayer 205® (antrypol®; suramin) is used intravenously for both *T. gambiense* and *T. rhodesiense* infection.

Tryparsamide is indicated in late *T. gambiense* infections with cerebrospinal fluid changes. Serious side effects are possible: interrupt treatment if visual disturbances appear. Melarsen® and its derivatives are useful in tryparsamide-resistant infections. Prognosis in advanced *T. rhodesiense* infection is poor with any treatment. Mel B and arsobal® have been used recently in both types of infections but need evaluation.

Pentamidine is effective in early *T. gambiense* infections; may be combined with tryparsamide; has not replaced Bayer 205® in treatment of *T. rhodesiense* infections.

C. Epidemic measures: Mass survey and treatment with tsetse control should be instituted as a matter of urgency. If epidemics continue to recur in the same area in spite of control measures, it may be necessary to move villages from sources of infection to a safer district. Other measures as in 9A.

D. International measures: Cooperative efforts of governments in endemic areas should be promoted. An International Bureau of Trypanosomiasis, acting as a clearing center for information, exists in Brazzaville, French Equatorial Africa.

TRYPANOSOMIASIS, AMERICAN

1. Identification—An infectious disease caused by a protozoon of the genus *Trypanosoma* occurring in the trypanosome form in the peripheral blood of man and of other vertebrate hosts, and in the leishmania form in endothelial and tissue cells. The acute stage, lasting several weeks, is characterized by fever, malaise, enlargement of spleen and liver, and myocardial damage. Other early signs are unilateral edema of the eyelids extending to the face with red-purple discoloration of the skin, inflammation and swelling of the lachrymal glands, conjunctivitis, and regional lymphadenopathy. The disease may become chronic with encephalopathy, or end with gradual myocardial failure; death is uncommon. Many infected persons, especially adults, have few or no clinical manifestations. Synonym: Chagas Disease.

Confirmation of diagnosis depends upon demonstration of trypanosome forms of the parasite in peripheral blood during febrile episodes, or of leishmania forms by muscle biopsy; by culture, by inoculation of white rats or guinea pigs, or by xenodiagnosis (feeding noninfected triatomid bugs on the patient and finding characteristic trypanosomes in the gut). A complement-fixation test provides presumptive evidence.

Two other species of trypanosome, *T. rangeli* and *T. ariarii*, have been found in blood films of man in Central and South American countries. They produce no demonstrable clinical disease, and are distinguishable from *T. cruzi* by morphological criteria.

2. Occurrence—The disease is confined to the Western Hemisphere, with a wide geographic distribution in rural areas of Mexico and Central and South America, in some areas being highly endemic. Endemic human infection has been reported from the United States (Texas). In several countries of the Americas naturally infected triatomids have been found outside areas where human cases have been recognized. Several species of *Triatoma* have been demonstrated as carriers of *Trypanosoma cruzi*.

in Texas, New Mexico, Arizona, and California. Infected wild rodents and opossums have been found in these areas, and raccoons in Maryland.

3. **Infectious agent**—*Trypanosoma cruzi*, a hemoflagellate.
4. **Reservoir and source of infection**—Reservoirs include infected persons and a number of domestic and wild animals, such as dog, cat, domestic pig, bat, house rat, wood rat, fox, ferret, monkey, opossum, raccoon, and armadillo. Source of infection is a triatomid bug.
5. **Mode of transmission**—By the fecal material of infected vectors; various blood-sucking species of *Reduviidae* (cone-nosed bugs), especially of *Triatoma*, *Rhodnius*, and *Panstrongylus* which frequently attack man. Infection may take place through conjunctivae, mucous membranes, abrasions, or wounds of the skin. Probably not transmitted by the actual act of biting; transmission by blood transfusion has been noted.
6. **Incubation period**—About 7 to 14 days.
7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Organisms are present in the blood only during the acute febrile period; the vector becomes infective 8 to 10 days after biting an infected host and remains infective for as long as 2 years.
8. **Susceptibility and resistance**—Children, especially infants under 2 years of age, are particularly susceptible.
9. **Methods of control**—

A. Preventive measures:

1. Construction or repair of dwellings to eliminate hiding places for the insect vector and shelter for the wild hosts.
2. Elimination of infected domestic animals and destruction of wild hosts in known endemic areas.
3. Use of a bed net in houses infested by the vector.
4. Systematic attack upon vectors through use of effective insecticides with residual action, such as gammexane (benzene hexachloride) and dieldrin. Residual spraying with DDT is not effective.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; not a reportable disease in most countries, Class 3B (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: None.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Search of bedding and rooms for the vector, and investigation among domestic and wild animals for evidence of infection. Other members of the family should be examined for infection.
8. Specific treatment: No method has recognized value. Bayer 7612R (ac), a 4-aminoquinoline, gives good results in some cases with little or no effect in others.

C. Epidemic measures: In areas of high incidence, field surveys to determine distribution and frequency of vectors.

D. International measures: None.

TUBERCULOSIS

1. Identification—A chronic bacterial disease and an important cause of death in most parts of the world. Primary infection usually goes unnoticed clinically; some patients have fever, vague constitutional symptoms, or roentgenographic evidence of infiltration of the lungs and enlarged tracheobronchial nodes. Tuberculin sensitivity appears within a few weeks after infection. The course of events thereafter is subject to much variation. Pleurisy with effusion may occur; in some areas, notably the Scandinavian countries, erythema nodosum is common; some few individuals develop disseminated tuberculosis. Such developments are more likely within the first 6 to 12 months. Usually the lesions heal spontaneously, leaving no residual changes except tuberculin sensitivity and occasionally pulmonary or tracheobronchial node calcifications. Disseminated or extrapulmonary (particularly skeletal) tuberculosis develops more often in infants and in Negroes. Late childhood primary disease is likely to be benign, and in young adults, it may resemble or merge into pulmonary tuberculosis.

Pulmonary tuberculosis generally has a chronic variable course, with exacerbations and remissions, but capable of arrest or relapse at any stage. Three stages (minimal, moderately advanced, and far advanced) are distinguished according to extent of lung involvement; activity is determined by progression or retrogression as detected in serial roentgenograms, by presence of tubercle bacilli, and by symptoms. Abnormal roentgen shadows indicative of pulmonary infiltration, cavitation, or fibrosis are common in advance of clinical manifestations. Symptoms of cough, fatigue, fever, weight loss, hoarseness, chest pain and hemoptysis, and physical signs of dullness and rales may occur, especially in advanced disease.

Specific diagnosis is by demonstration of tubercle bacilli in sputum by stained smear, concentration and culture, or animal inoculation. Negative results on microscopic examination of sputum do not rule out tuberculosis; repeated examinations with more sensitive methods are eventually successful in most active cases; gastric washings or laryngeal swabs should be examined when sputum is absent or negative. Tuberculin test is positive in active tuberculosis except in critically ill persons, during certain intercurrent diseases (e.g., measles), and sometimes in old age. Negative reaction therefore aids differential diagnosis.

Extrapulmonary tuberculosis is usually a result of hematogenous dissemination of tubercle bacilli during the primary phase, as miliary tuberculosis, tuberculosis of bones and joints, central nervous system (tuberculous meningitis), lymphatic glands, and kidneys; or as a complication of pulmonary tuberculosis involving intestines or larynx. Diagnosis is by isolation of tubercle bacilli from a lesion or exudate (cerebrospinal fluid in meningitis) or by histopathology. In the United States these forms are far less common than pulmonary tuberculosis, and as causes of death are decreasing more rapidly.

2. Occurrence—Present in nearly every community but frequency varies widely. In some countries the trend of mortality has been steadily downward for many years, but tuberculosis remains one of the most serious communicable diseases of man. Mortality rates range in different countries from about 5 to 100 deaths per 100,000 population per year. They reflect in part the social and economic welfare of a region. In the United States mortality is much higher in Negroes than whites. Epidemics have been reported among school children in crowded classrooms or other groups congregated in enclosed spaces.

Reported case incidence and prevalence are declining more slowly than mortality, owing to improved case finding and better survivorship of patients treated with antimicrobial therapy.

Photofluorographic surveys show prevalence of pulmonary tuberculosis to be low under age 15 and rising gradually with age, although a large proportion of lesions of older persons are inactive. At present, fewer than 1 in 1,000 persons examined in such surveys in the United States is found to have previously unknown active tuberculosis. A striking shift is noted toward higher relative frequency and mortality in males past middle age, consequent upon the faster decline in other age-sex groups. Prevalence of disease as estimated by roentgenographic survey is apt to be no higher in Negroes, owing to the more chronic course of the disease and higher frequency of inactive lesions in whites.

The prevalence of infection, as manifested by reaction to a small dose of tuberculin, increases with age and is usually higher in cities than in rural areas. In many places there has been a rapid decline in prevalence of infection in recent decades, and in the United States fewer than 10% of persons aged 17 to 20 years now react positively to 5 TU (tuberculin units) of PPD (purified protein derivative).

Bovine type tuberculosis in man is rare in the United States, Canada, Scandinavia, and the Netherlands. It is rapidly being eliminated in Great Britain, Switzerland and parts of France and Germany. The bovine type disease remains a problem in South America, southern Europe, parts of Africa, Asia, and Australia.

3. **Infectious agent**—*Mycobacterium tuberculosis*, tubercle bacillus. The human type causes nearly all pulmonary tuberculosis and the bovine type a considerable share of extrapulmonary disease, proportions varying according to opportunities for infection.
4. **Reservoir and source of infection**—Reservoir is primarily man; in some areas, also diseased cattle. Source of infection is respiratory secretions of persons with "open" (bacillary-positive) pulmonary tuberculosis; milk from tuberculous cattle. Patients with extrapulmonary tuberculosis are usually not responsible for infection of others.
5. **Mode of transmission**—Coughing or sneezing by patients with open pulmonary tuberculosis sets up a cloud of infectious material; minute particles may be inhaled directly or after settling and resuspension as dust. Direct and indirect contact are important; alimentary infection, as by contaminated eating and drinking utensils, is less so. Infection usually results from the continued and intimate exposure that characterizes household relationships; some family contacts avoid infection for long periods.
Bovine tuberculosis is transmitted by ingestion of unpasteurized milk or dairy products from tuberculous cows, by air-borne infection in barns and by handling contaminated animal products.
6. **Incubation period**—From infection to demonstrable primary phase lesions, about 4 to 6 weeks; from infection to progressive pulmonary or extrapulmonary tuberculosis may be years, with the first 6 to 12 months most hazardous.
7. **Period of communicability**—As long as tubercle bacilli are being discharged by the patient. Commences when a lesion becomes open (discharges tubercle bacilli) and continues until healed or death occurs. Some patients remain sputum-positive intermittently for years. Degree of communicability depends upon numbers of bacilli discharged, cough-

ing habits, and hygienic practices of the patient. Antimicrobial therapy, pulmonary resection and collapse therapy commonly shorten communicability.

8. Susceptibility and resistance—Susceptibility is general; highest in children under 3 years, lowest from 3 to 12 years, and intermediate thereafter; greater in aboriginal races than in populations long exposed to the disease; in the undernourished, neglected, and fatigued more than in the well fed and well cared for. Persons with silicosis or diabetes are especially susceptible. The resistance conferred by healed primary infection is difficult to assess, but is not complete. Relapse of long latent infection particularly in older males accounts for a large proportion of active cases.

9. Methods of control—

A. Preventive measures:

1. Education of the public in the danger of tuberculosis, its mode of spread, and methods of control.
2. Provision of adequate hospital facilities for isolation and treatment of active cases.
3. Provision of roentgenographic and clinical facilities for examination of contacts and suspects, and for clinical supervision and treatment of ambulant patients and those not hospitalized.
4. Public health nursing service for home supervision of patients and to encourage and arrange for examination of contacts.
5. Insuring a safe milk supply through pasteurization of milk and elimination of tuberculosis among dairy cattle.
6. Measures to avoid inhalation of dangerous concentrations of silica dust in industrial plants and mines.
7. Routine roentgenographic examination of groups that have a higher prevalence of tuberculosis than the general population, such as nurses, medical students, patients and outpatients in general and mental hospitals, and selected groups of industrial workers; also those who constitute a special hazard to others if infected, such as school personnel. When feasible, initial screening by tuberculin test may be substituted with examination by roentgen ray restricted to reactors.
8. Roentgenographic screening of adult populations in communities where the frequency of tuberculosis is known to be excessive.
9. Tuberculin-testing surveys, employing 5 TU of PPD intracutaneously, may be helpful in identifying areas or groups at high risk of infection. Large doses of tuberculin should not be employed; non-specific reactions may follow.
10. BCG vaccination of uninfected persons. Vaccination confers definite, though partial, protection. In places and among population groups where risk of infection is low, as presently in most parts of the United States, vaccination has little role in tuberculosis control. May be used for household contacts of active cases and for persons exposed to infection by reason of occupation, such as medical and nursing students. In areas of high mortality where economic resources do not permit other control measures to be widely practiced, such as hospitalization of cases, mass vaccination of nonreactors is feasible and gives hope of partial control. Preliminary tuberculin testing is unnecessary for newborn infants. Intracutaneous vaccina-

tion is best, and potent vaccine essential. Proper vaccination induces tuberculin sensitivity in over 90% of individuals, but duration is variable. BCG must not be administered to tuberculin positive individuals.

11. Chemoprophylactic isoniazid (INH) management of population groups having unusually high rates is under investigation.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory case report in most states and countries, Class 2B (p. 5). Health departments should maintain a current register of active cases.
2. Isolation: A period of hospital or sanatorium treatment removes a focus of infection from the home, teaches the patient hygienic essentials of tuberculosis control, and increases the chances of recovery. For patients remaining at home, public health nursing supervision, including instruction in personal hygiene; compulsory isolation of those with open tuberculosis who do not observe necessary precautions is occasionally required.
3. Concurrent disinfection: Of sputum and articles soiled therewith, including handkerchiefs, cloth or paper napkins, and of eating utensils used by patient. Patients should be trained to cover mouth and nose in coughing and sneezing.
4. Terminal disinfection: Wet cleaning of walls and floors and subsequent exposure to sunlight and fresh air.
5. Quarantine: None.
6. Immunization of contacts: BCG vaccination of tuberculin negative contacts may be warranted. (See Section 9A10.)
7. Investigation of contacts and source of infection: Roentgenologic examination of all members of the household of a newly discovered case and all intimate extrahousehold contacts, with particular attention to adult contacts. Alternatively, tuberculin testing may be practiced, with subsequent roentgenologic examination of reactors. Annual retesting of tuberculin-negative persons, with intensive study of converters and their contacts, has been productive in some situations.
8. Specific treatment: Most primary infections heal without treatment; if recognized in an active stage in susceptible age and racial groups, antimicrobial therapy should be given for a full year. Chemoprophylaxis for young infected children is under study; initial reports are encouraging.

Patients with active pulmonary tuberculosis should be in a hospital or on a hospital regimen of rest followed by graduated resumption of activity. Prompt treatment with antimicrobial drugs is indicated as soon as cultures have been obtained for diagnosis and sensitivity tests. Two drugs in combination are presently employed. The most effective combination is isoniazid (INH) and para-aminosalicylic acid (PAS) given orally for 18 months. Streptomycin (SM), given intramuscularly, is usually reserved for patients who cannot tolerate PAS, or the few who become drug fast to INH. Damage of the eighth cranial nerve by SM is rare when used no oftener than 3 times weekly; PAS gives rise in most patients to mild digestive disturbances. Where laboratory facilities permit, initial cultures of tubercle bacilli should be examined for drug sensitivity, to be repeated in 6 months if the sputum fails to

become negative or reverts to positive after a series of negatives. Drug fastness necessitates the addition or substitution of another antimicrobial agent.

Reversible forms of lung collapse (pneumothorax, pneumoperitoneum and phrenic nerve interruption) have largely been abandoned. Pulmonary resection is used increasingly in conditions of permanent and irreversible damage; this should be undertaken within 6 to 9 months from initiation of antimicrobial therapy. Thoracoplasty is still used occasionally in selected cases.

In extrapulmonary tuberculosis, antimicrobial therapy for 18 months as described above is combined with specific measures, often surgical, suited to the particular form and type of the disease.

In miliary and meningeal tuberculosis, treatment should be started immediately with INH in full dosage by mouth and daily intramuscular injections of SM. Intrathecal SM largely has been abandoned. After 3 months of treatment with INH and SM, INH and PAS are usually substituted for another 15 months.

C. Epidemic measures:

1. Alertness to recognize aggregations of new cases resulting from contact with an unrecognized infectious case; intensive search for the source of infection in such situations.

D. International measures: Roentgenographic screening of individuals prior to emigration to detect communicable and potentially communicable tuberculosis.

TULAREMIA

1. **Identification**—An infectious disease of wild animals and man; onset is with chills and fever, the patient usually prostrated and confined to bed. Frequently a local ulcer appears at the site of the original infection and lymph nodes draining this area become swollen and tender, and commonly suppurate. Fatality in untreated cases is about 5%; with treatment, negligible.

Diagnosis is confirmed by inoculation of animals with material from local lesions or with sputum, by isolation of the microorganism bacteriologically, and by agglutination reaction.

2. **Occurrence**—Throughout North America, in many parts of continental Europe, Russia, and in Japan. Occurs in the United States in every month of the year, but especially during rabbit hunting season in autumn.
3. **Infectious agent**—*Pasteurella tularensis* (Bacterium tularensis).
4. **Reservoir and source of infection**—Reservoirs are wild animals and some domestic animals; wild rabbits and hares, woodchuck, coyote, muskrat, opossum, tree squirrel, quail, skunk, water rat of Europe (*Arvicola amphibus*), cat, deer, dog, fox, hog, sage hen, sheep, and bull snake; also woodticks. The source of infection is the blood and tissues of infected animal hosts or an infective arthropod.
5. **Mode of transmission**—By bite of infected flies or ticks, and by inoculation of skin or conjunctival sac through handling infected animals, as in skinning, dressing, or performing necropsies, or by fluids from infected flies, ticks, rabbits, and woodchucks. Arthropods directly related to disease in man are the following: one species of deer fly, *Chrysops*

disalis; the wood tick, *Dermacentor andersoni*; the dog tick, *Dermacentor variabilis*; and the Lone Star tick, *Amblyomma americanum* and in Sweden the mosquito, *Aedes cinereus*. By ingestion of insufficiently cooked rabbit meat and drinking contaminated water. Rare cases occur from bites of coyotes, skunks, hogs, cats, and dogs, where the mouth of the animal was presumably contaminated from eating infected rabbits. Laboratory infections are relatively frequent.

6. Incubation period—From 24 hours to 10 days, usually 3 days.

7. Period of communicability—Under natural conditions not directly transmissible from man to man. The infectious agent may be found in the blood of man during the first 2 weeks of the disease, and in lesions of the disease up to a month from onset and sometimes longer. Flies are infective for 14 days, ticks throughout lifetime, about 2 years. Refrigerated rabbits kept constantly frozen at -15°C (5°F) may remain infective for more than 3 years.

8. Susceptibility and resistance—All ages are susceptible; permanent immunity follows recovery, except that through abrasion of the hand or by contact with contaminated material an immune person may acquire a local tularemic papule which harbors virulent organisms but causes no notable constitutional reaction.

9. Methods of control—

A. Preventive measures:

1. Avoid bites of flies and ticks or handling such arthropods when working in endemic areas.
2. The use of rubber gloves by persons engaged in dressing wild rabbits or conducting laboratory experiments. Thorough cooking of meat of wild rabbits.
3. Avoid drinking raw water in areas where the disease prevails among wild animals.
4. Killed vaccines are of limited value. Vaccines made from living attenuated strains may prove useful, but are not immediately available and seldom indicated.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas (USA); in many countries not a reportable disease, Class 3A (pp. 5, 6).
2. Isolation: None.
3. Concurrent disinfection: Of discharges from ulcer, lymph nodes, or conjunctival sac.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Not indicated.
7. Investigation of contacts and source of infection: Should be undertaken in each case.
8. Specific treatment: Streptomycin, the tetracyclines, and chloramphenicol are effective when continued until temperature is normal for 4 to 5 days.

C. Epidemic measures: Search for sources of infection related to arthropods, to animal hosts, and to water. Control measures as thus indicated (see 9A). Interstate or interarea shipment of infected animals or carcasses should be prohibited.

D. International measures: None.

TYPHOID FEVER

1. **Identification**—A systemic infection characterized by continued fever, involvement of lymphoid tissues, especially ulceration of Peyer's patches, enlargement of spleen, rose spots on trunk, and constipation more commonly than diarrhea. Many mild, atypical infections remain unrecognized. A usual fatality of 10% is reduced to 2 to 3% by antibiotic therapy. Synonyms: Enteric Fever, Typhus Abdominalis.

Typhoid bacilli are found in blood during first 2 weeks and in feces and urine after second week. Widal reaction becomes positive during second week; O agglutinins more significant than H agglutinins.

2. **Occurrence**—Widespread throughout world. Endemic in some rural areas of United States, but commonly occurring as sporadic cases and as small contact and carrier epidemics; steadily falling in incidence, particularly in urban areas. Still common in many countries of the Far East, Middle East, eastern Europe, Central and South America, and in Africa.
3. **Infectious agent**—*Salmonella typhi*, typhoid bacillus. About 50 types can be distinguished by Vi-phage.
4. **Reservoir and source of infection**—Reservoir is man; patients and carriers. The source of infection is feces and urine of infected persons. Family contacts may be transient carriers; fecal carriers more common than urinary. The carrier state is most common among persons over 40 years of age, especially females; fecal carriers frequently have a typhoid cholecystitis, usually permanent unless cholecystectomy is done.
5. **Mode of transmission**—Direct or indirect contact with patient or carrier. Principal vehicles of spread are contaminated water and food. Raw fruits and vegetables are important factors in some parts of the world; milk, milk products, and shellfish in others. Contamination is usually by hands of carrier or missed case. Under some conditions, flies are vectors.
6. **Incubation period**—Variable; average 2 weeks, usual range 1 to 3 weeks.
7. **Period of communicability**—As long as typhoid bacilli appear in excreta; usually from second week throughout convalescence, thereafter variable. About 10% of patients will discharge bacilli 3 months after onset; 2 to 5% become permanent carriers.
8. **Susceptibility and resistance**—Susceptibility is general, although many adults appear to acquire immunity through unrecognized infections; attack rates decline with age after second or third decades. A high degree of resistance usually follows recovery. The degree of artificial active immunity conferred by typhoid vaccine is uncertain.

9. Methods of control—

A. Preventive measures:

1. Protection, purification and chlorination of public water supplies; construction of safe private supplies. For individual or small group protection and while traveling or in the field, water is preferably boiled, a rolling boil for 2 to 5 minutes; or chlorine (halazone) or iodine (globaline) disinfecting tablets can be added directly to water in number dependent upon turbidity and amount of water to be treated. The iodine preparation has the advantage that it kills amebic cysts as well as bacteria.
2. Sanitary disposal of human excreta.
3. Boiling of milk or pasteurization of milk and dairy products, including cheese. Sanitary supervision of commercial milk production,

transport, processing, and delivery. Proper storage and refrigeration in stores and homes.

4. Limitation of collection and marketing of shellfish to those from approved sources.
 5. Sanitary supervision of processing, preparation and serving of all foods, especially those that are eaten raw; special attention to provision and use of hand washing facilities. Protection of foods against flies by screening.
 6. Fly control by screening, by fly traps, and by contact poisons and space sprays. Control of fly breeding by provision for adequate garbage collection; elimination of garbage dumps and substitution of land-fill operations; provision for proper disposal of feces.
 7. Immunization with a vaccine of high antigenicity. Current practice is to vaccinate persons subject to unusual exposure through occupation or travel, those living in areas of high endemic incidence, and institutional populations in which maintenance of sanitation is difficult. Periodic reinforcing injections are desirable, commonly once in 3 years.
 8. Discovery and supervision of typhoid carriers. Those of duration exceeding 1 year may be released from supervision and restriction of occupation (9A9) only after 6 consecutive negative cultures of authenticated specimens of feces and urine taken 1 month apart (cf. 9B2). Cholecystectomy usually ends the carrier state.
 9. Instruction of convalescents and chronic carriers in personal hygiene, particularly as to sanitary disposal of excreta, handwashing after defecation and before eating, and exclusion from acting as food handlers.
 10. Education of general public and particularly of food handlers concerning sources of infection and modes of transmission.
- B. Control of patient, contacts, and the immediate environment:**
1. Report to local health authority: Obligatory case report in most states and countries, Class 2A (p. 5).
 2. Isolation: In flyproof room. Hospital care desirable for patients who cannot command adequate sanitary environment and nursing care at home. Release from supervision by local health authority should be by not less than 3 negative cultures of feces and urine, taken at least 24 hours apart and not earlier than 1 month after onset; if any one of this series is positive, not less than 3 negative cultures at intervals of 1 month and within the 12 months following onset (cf. 9A8).
 3. Concurrent disinfection: Of feces and urine and articles soiled therewith. In communities with modern and adequate sewage disposal systems, feces and urine can be disposed of directly into sewer without preliminary disinfection.
 4. Terminal disinfection: Cleaning.
 5. Quarantine: Family contacts should not be employed as food handlers during period of contact nor before repeated negative feces and urine cultures are obtained.
 6. Immunization of contacts: Administration of typhoid vaccine to family, household, and nursing contacts who have been or may be exposed to cases or carriers.
 7. Investigation of contacts and source of infection: Actual or probable source of infection of every case should be determined by search

for common and individual sources, unreported cases and carriers, or contaminated food, water, milk or shellfish. Presence of Vi-agglutinins in blood of suspected carriers is suggestive of the carrier state. Organisms from patients and carriers should be phage-typed to determine occurrence of same type, indicating thereby a presumed common origin.

8. Specific treatment: Chloramphenicol; an initial oral loading dose is followed by oral doses every 6 hours until temperature is normal, then in smaller doses for total of 14 days. If chloramphenicol is contraindicated or ineffective, substitute a tetracycline antibiotic promptly.

C. Epidemic measures:

1. Intensive search for case or carrier who is source of infection.
2. Exclusion of suspected food.
3. Boiling of milk or pasteurization or exclusion of suspected milk supplies or other foods suspected on epidemiologic evidence, pending elimination of cause of contamination.
4. Chlorination under competent supervision of suspected water supply or its exclusion. All water used for drinking must be chlorinated or boiled before use.

D. International measures: Inoculation with triple typhoid vaccine (TAB, typhoid, paratyphoid A, paratyphoid B) is advised for international travelers unless protection is afforded through previous attack; not a legal requirement of any country. The risk is minimal in the United States, Canada, Great Britain and northwest Europe.

TYPHUS FEVER, ENDEMIC FLEA-BORNE

1. **Identification**—The clinical course resembles that of epidemic louse-borne typhus (p. 212) but tends to be milder. Fatality for all ages is about 2% with prognosis grave in older people. Synonym: Murine Typhus. The Weil-Felix reaction is usually positive with *Proteus* OX19 after the ninth day; the complement-fixation reaction a few days later. Differential diagnosis from louse-borne typhus is by serologic tests using rickettsial suspensions.
2. **Occurrence**—World-wide in areas where men and rats occupy the same buildings. Formerly several thousand cases occurred each year in the United States; the current level is about 100 cases, mainly in Gulf and South Atlantic seaboard states and during summer months when fleas are abundant. Changes in agricultural practices and increased rat populations of farms make the disease primarily rural, replacing the former urban distribution centered around feed and grain stores.
3. **Infectious agent**—*Rickettsia prowazeki*, var. *typhi* (*Rickettsia mooseri*).
4. **Reservoir and source of infection**—The infection is maintained in nature by a rat-flea-rat cycle. Rats are the reservoir, commonly *Rattus rattus* and *Rattus norvegicus*. The immediate source of infection is an infective rat flea, commonly *Xenopsylla cheopis*.
5. **Mode of transmission**—Infected fleas excrete rickettsiae in feces, defecate after sucking blood, and contaminate the fresh skin wound. Inhalation of dried infective flea feces may account for an occasional case.
6. **Incubation period**—From 6 to 14 days, commonly 12 days.

7. **Period of communicability**—Under natural conditions not directly transmissible from man to man. Fleas remain infective for life, which ordinarily is as long as that of uninfected fleas.
8. **Susceptibility and resistance**—Susceptibility is general. One attack confers immunity, not always permanent.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Application of insecticide powders with residual activity (10% DDT or other compounds) to rat runs, burrows, and harborages.
 2. Rodent control measures should be delayed until flea populations have been reduced by insecticides, to avoid temporary increase in cases.
 3. Development and testing of a murine typhus vaccine has been abandoned because of the effectiveness of modern insecticides and the antibiotic management of patients. The need for general protection of populations no longer exists.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Case report obligatory in most states and countries, Class 2B (p. 5).
 2. Isolation: None.
 3. Concurrent disinfection: None.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Search for rodents around premises or home of patient.
 8. Specific treatment: As for epidemic typhus; see 9B8 (p. 214).
 - C. **Epidemic measures:** In endemic areas with numerous cases, widespread use of DDT has markedly reduced the flea index of rats, and incidence of infection in rats and man.
 - D. **International measures:** None.

TYPHUS FEVER, EPIDEMIC LOUSE-BORNE

1. **Identification**—A rickettsial disease with a history of great epidemics and a continued existence in numerous areas of the world. The onset is variable, often sudden and marked by headache, chills, fever, and general pains; a macular eruption appears on the fifth or sixth day, toxemia is usually pronounced and the disease terminates in rapid lysis after about 2 weeks of fever. In the absence of specific therapy the fatality varies from 10 to 40% in different epidemics, and increases with age. Mild infections may occur with eruption evanescent or absent, especially in vaccinated persons. A recrudescence of epidemic typhus fever may occur years after the primary attack (Brill's disease); it differs from the classical type in that it need not be associated with lousiness and is milder, with fewer complications and lower fatality. Synonyms: Typhus Exanthematicus, Classical Typhus Fever.
 The Weil-Felix reaction with Proteus OX19 is usually positive with scrums obtained after the tenth day, the complement fixation test a few days later.

2. **Occurrence**—In most colder areas of the world where appreciable groups of people live under unhygienic conditions and are louse infested. Endemic centers exist in mountainous regions of Mexico and Central and South America, the Balkans and eastern Europe, North Africa and mountainous areas of Central and South Africa, and in numerous countries of Asia. Prior to modern methods of control, epidemics were frequent among military and refugee populations and in areas suffering famine or war.
3. **Infectious agent**—*Rickettsia prowazeki*, var. *prowazeki*.
4. **Reservoir and source of infection**—Man is the reservoir and responsible for maintaining the infection during interepidemic periods. The immediate source of infection is lice infected by feeding on the blood of a patient with febrile typhus fever. Patients with recrudescent typhus (Brill's disease) can infect lice and probably serve as foci for new outbreaks in louse-infested communities.
5. **Mode of transmission**—Infected body lice, *Pediculus humanus*, excrete rickettsiae in their feces and usually defecate at the time of feeding. Man is infected by rubbing feces or crushed lice into the wound made by the bite or into superficial abrasions of the skin. Inhalation of dried infective louse feces as dust from dirty clothes may account for some infections.
6. **Incubation period**—From 6 to 15 days, commonly 12 days.
7. **Period of communicability**—Patients are infective for lice during the febrile illness and possibly for 2 or 3 days after the temperature returns to normal. The living louse is infective as soon as it begins to pass rickettsiae in feces; earlier if crushed. The louse is regularly killed by the infection. Rickettsiae may remain viable in the dead louse for weeks. Under natural conditions the disease is not directly transmissible from man to man.
8. **Susceptibility and resistance**—Susceptibility is general. The disease in children and in vaccinated adults is mild and may go unrecognized. Attack usually confers permanent immunity.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Application by hand or power blower of residual insecticide powder (10% DDT or newer lousicides) at appropriate intervals to clothes and persons of populations living under conditions favoring lousiness. Lice are known to develop DDT resistance, in which case lindane may be substituted as a dusting powder.
 2. Improvement of living conditions with provision for frequent bathing and washing of clothing.
 3. Individual prophylaxis of persons subject to unusual risk through insecticide applied at appropriate intervals to clothing by dusting or by impregnation.
 4. Immunization of susceptible persons or groups of persons entering typhus areas, particularly military or labor forces, or of residents at unusual risk. The usual vaccine contains rickettsiae grown in yolk sac of developing chick embryo and inactivated by formalin. The vaccine is administered in 2 doses of 1 ml. each at an interval of not less than 7 days, and a third dose of 0.5 ml. not less than 6 months after the second dose. A booster inoculation of 0.5 ml. at yearly intervals thereafter is necessary where typhus is a real and persisting hazard. For vaccinated persons the risk of illness is reduced, the course of the disease modified, and the fatality lowered.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report universally required by international regulation, Class 1 (p. 5).
2. Isolation: Not required after proper delousing of patient, clothing, living quarters, and household contacts.
3. Concurrent disinfection: Appropriate insecticide powder applied to clothing and bedding of patient and contacts; treatment of hair for louse eggs (nits) with tested chemical agents.
4. Terminal disinfection: If death occurs before delousing, thorough application of insecticides to body and clothing.
5. Quarantine: Louse-infested susceptibles exposed to typhus fever should be quarantined for 15 days but may be released directly after application of insecticide with residual effect.
6. Immunization of contacts: All immediate contacts.
7. Investigation of contacts and source of infection: Every effort should be made to trace the source of infection to direct or indirect contact with a preceding case.
8. Specific treatment: The tetracyclines or chloramphenicol orally in a loading dose followed by daily doses until the patient becomes afebrile (usually 2 days) and for one additional day.

C. Epidemic measures:

1. Delousing: The most important measure for the rapid control of typhus, where reporting has been good and numbers of cases are small, is application of insecticides with residual effect to all contacts. Where infection is known to be widespread, systematic application of residual insecticide to all persons in the community is indicated.
2. Immunization: Of persons in contact with cases; vaccination may be offered to entire community.

D. International measures:

1. Telegraphic notification to WHO and to adjacent countries, by governments, of the existence of an epidemic of typhus fever.
2. Measures applicable to ships, aircraft, and land transport arriving from typhus areas are specified by International Sanitary Regulations, World Health Organization, annotated edition, Geneva, 1957.
3. International travelers: Infected persons or suspects are not permitted to depart a country until demonstrated free of infection. No country currently requires immunization against typhus for entry. Vaccination is recommended for all persons entering areas where typhus is present. Health authorities may require disinfection before departure of healthy travelers considered liable to spread typhus, and similarly at ports of arrival.

UPPER RESPIRATORY DISEASE, ACUTE**(Adenovirus and Unknown Etiology)**

Acute upper respiratory illnesses of known or presumed viral etiology are perhaps the most common diseases of man. Their importance lies in the great frequency of their occurrence, with attending discomfort, disability and complications. The illnesses generally last from 2 to 5 days and consist of a variety of manifestations ranging from afebrile coryza to more severe febrile syndromes.

Development of improved tissue culture technics has resulted in the isolation of many new viruses from persons with these diseases. The adenoviruses are clearly related to acute febrile pharyngitis, pharyngoconjunctival fever and acute respiratory disease (ARD) or catarrhal fever of military recruits. The significance of other new viruses such as the JH, the 2060 agent, the Eaton virus, the "croup associated" viruses, the parainfluenza viruses and the hemadsorption or HA viruses is not adequately determined. In all, it appears probable that presently known viruses cause only a small proportion of acute febrile respiratory illnesses and their role in the afebrile illnesses remains undetermined. The adenovirus syndromes are here described, together with brief consideration of acute febrile respiratory diseases of unknown etiology. A description of the common cold, also of unknown etiology, then follows.

A. PHARYNGOCONJUNCTIVAL FEVER

1. **Identification**—An acute respiratory illness characterized by gradual onset of fever, irritated throat, headache, malaise, and conjunctivitis which may occur singly, together, or in any combination. Chilliness, anorexia, minimal hoarseness, and slight cough also may occur. The conjunctivitis is follicular, nonpurulent, and unilateral or bilateral; commonly a prominent feature of summer outbreaks associated with swimming, otherwise less frequent, mild and of short duration. The posterior pharynx may be moderately red and commonly studded with prominent lymph follicles; a grayish white exudate may or may not be present. Leucocyte count and distribution, and the bacterial flora of the throat, are usually within normal limits. Complete recovery within 4 to 5 days is the rule, but pneumonia, rarely fatal, has been recognized. Synonyms: Nonbacterial Pharyngitis, Nonbacterial Exudative Tonsillitis and Pharyngitis, and Catarrhal Conjunctivitis.

Diagnosis may be confirmed by virus isolation and serologic studies.

2. **Occurrence**—Serologic evidence indicates that infection with various adenovirus types is world-wide. Outbreaks of clinical disease have been reported in Europe, North America, and Japan. The disease occurs both endemically and in epidemics, the latter principally in institutions for children and summer camps, where 70% of children may be attacked within a few weeks. Although adenovirus infections perhaps account for less than 3% of all respiratory illness in civilian populations, these infections appear to be highly prevalent in young children of densely populated areas.
3. **Infectious agents**—Adenoviruses of several types, with Type 3 predominating in both epidemic and sporadic cases.
4. **Reservoir and source of infection**—Reservoir is man, primarily patients; inapparent infection occurs but the importance of carriers is undetermined. The source of infection is discharges from the nose, throat, conjunctiva, and intestinal tract of infected persons.
5. **Mode of transmission**—Principally by intimate or direct contact or by droplet spread, although indirect contact with contaminated articles may occur. Summer outbreaks have been associated with both well and poorly chlorinated swimming pools but there is no evidence incriminating water as a vehicle of transmission.
6. **Incubation period**—Between 5 and 7 days.
7. **Period of communicability**—During the latter part of incubation and through the acute illness; approximately 10 days.
8. **Susceptibility and resistance**—Susceptibility is apparently universal, although immunity presumably follows infection with a specific type

virus. Resistance increases with age, probably due to specific immunity from previous infections.

9. Methods of control—

A. Preventive measures:

1. Adenovirus vaccines containing Types 3, 4, and 7 are effective in preventing disease caused by these types in volunteers and in military recruits. Adequate doses of a vaccine containing Type 3 would presumably protect children and may be useful for special groups as in institutions or summer camps when high attack rates are anticipated. Adenovirus vaccines are not recommended for general or widespread use in civilian populations.
2. Education in personal hygiene.
3. Avoidance of overcrowding in institutions caring for children.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6).
2. Isolation: Not of established value.
3. Concurrent disinfection: Disposal of nose and mouth discharges.
4. Terminal disinfection: Airing and sunning of room and bedding.
5. Quarantine: None.
6. Immunization of contacts: None.
7. Investigation of contacts and source of infection: Unprofitable.
8. Specific treatment: None.

C. *Epidemic measures*: None; available evidence suggests that immunization might be effective.

D. *International measures*: None.

B. ACUTE RESPIRATORY DISEASE (ARD) OF MILITARY RECRUITS

1. **Identification**—An acute grippé-like infection of the respiratory tract characterized by gradual onset, fever, chilliness, headache, malaise, anorexia, minimal nasal symptoms, irritated throat, minimal hoarseness and dry cough lasting usually from 4 to 7 days. Physical signs ordinarily are few, and consist most frequently of mild injection of the pharynx and palate and lymphoid hyperplasia on the pharyngeal wall. Evidence of pulmonary involvement may be found in approximately 10% of patients. Leucocyte counts are within normal limits and cultures of the throat, or of sputum if available, reveal only the normal bacterial flora of the respiratory tract. Recovery is complete without complications. Synonyms: Catarrhal Fever, Febrile Catarrh.
2. **Occurrence**—So far as is known, occurs almost exclusively as a disease of military recruits. Occurs both endemically and epidemically, epidemics being a feature of winter months in individual units such as a company; they begin 2 to 3 weeks after arrival of men on a post and run their course in the subsequent 3 to 4 weeks. Attack rates may be high; from 20 to 40% of recruits may require hospitalization and an equal proportion may be infected in less severe or inapparent manner, as indicated by serological studies. The true geographic distribution is unknown, but epidemics have been recognized in North America and Europe.
3. **Infectious agents**—Adenoviruses, predominantly Types 4 and 7, but occasionally Types 3 and 14.

4. **Reservoir and source of infection**—Reservoir is man; patients, and possibly carriers. The source of infection is discharges from the nose and mouth of infected persons.
5. **Mode of transmission**—Usual transmission is by intimate or direct contact or by droplet spread; indirect transmission has not been established.
6. **Incubation period**—From 5 to 6 days.
7. **Period of communicability**—From 5 to 7 days following onset of illness, as judged by isolation of the virus from the throat.
8. **Susceptibility and resistance**—Susceptibility is presumably universal. Infection is followed by the development of type-specific antibodies which persist for many years and presumably are associated with immunity. Epidemiological evidence suggests development of herd immunity to the predominant type or types after an epidemic.
9. **Methods of control**—
 - A. *Preventive measures:*
 1. Adenovirus vaccines containing Types 3, 4 and 7 are highly effective in preventing outbreaks in recruit populations; their use by the military is recommended.
 2. Education in personal hygiene.
 - B. *Control of patient, contacts, and the immediate environment:*
 1. Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (p. 6).
 2. Isolation: The isolation of patients has little or no effect on the spread of the epidemic disease in recruits, probably because of the high proportion of mild or inapparent infections.
 3. Concurrent disinfection: Disposal of nose and throat discharges.
 4. Terminal disinfection: Airing and sunning of room and bedding.
 5. Quarantine: None.
 6. Immunization of contacts: None.
 7. Investigation of contacts and source of infection: Unprofitable.
 8. Specific treatment: None.
 - C. *Epidemic measures:* The most effective measure for the control of epidemics is immunization with vaccine containing adenovirus Types 4 and 7. Gamma globulin produces transient immunity. Such procedures as isolation, ultraviolet irradiation, aerosols, and dust control have not been effective.
 - D. *International measures:* None.

C. ACUTE FEBRILE RESPIRATORY DISEASES OF UNKNOWN ETIOLOGY

The clinical manifestations of these diseases are similar to ARD or catarrhal fever of military recruits but more variable. A few of the known viruses and several others as yet unknown are likely responsible for these infections. Incubation periods vary from a few days to a week or longer. The human respiratory tract and its secretions appear to be the chief source and route of infection. These diseases occur at all ages and throughout the world. In temperate zones they are highly prevalent, particularly during cold seasons and predominantly in young children. No effective control measures are known; good personal hygiene and adequate medical care are recommended.

D. COMMON COLD

1. **Identification**—An acute catarrhal infection of the upper respiratory tract characterized by coryza, lacrimation, irritated nasopharynx, chilliness, and malaise lasting 2 to 7 days. Probably never fatal; importance rests in days of disability and in predisposing to more serious respiratory tract infections. Fever is uncommon, particularly in infants and adults. Complications of catarrhal sinusitis, otitis media, laryngitis, tracheitis, and bronchitis are frequent.
No confirmatory laboratory tests; leucocyte count and distribution are normal.
2. **Occurrence**—World-wide distribution; endemic and epidemic. In temperate zones, incidence rises during winter months. Most persons, except in small isolated communities, have 1 to 6 colds yearly. Incidence highest in children under 5 years; gradual decline with increasing age.
3. **Infectious agent**—The infection has been transmitted experimentally to chimpanzees and man by a filter-passing agent, presumably a virus. Cultivation and characterization not certainly achieved; more than one type is probable but undetermined. Role of bacteria in the usual case is uncertain; pyogenic bacteria of the respiratory tract (pneumococci, streptococci, *H. influenzae*) may cause suppurative complications. Some colds may be responses to toxic, allergic, or psychologic stimuli.
4. **Reservoir and source of infection**—Reservoir is man; source of infection is discharges from nose and mouth of infected persons.
5. **Mode of transmission**—Presumably transmission is by direct contact or by droplet spread; indirectly by handkerchiefs, eating utensils, or other articles freshly soiled by discharges of the infected person.
6. **Incubation period**—Between 12 and 72 hours, usually 24 hours.
7. **Period of communicability**—Nasal washings taken 24 hours before onset and for 5 days after onset produce symptoms experimentally in human volunteers.
8. **Susceptibility and resistance**—Susceptibility is universal. Inapparent and abortive attacks occur; frequency undetermined. Epidemiological evidence suggests limited and transient immunity; no immunity demonstrated in volunteers reinoculated after 3 weeks. Means for artificial immunization do not exist.
9. **Methods of control**—
 - A. **Preventive measures:**
 1. Education in the niceties of personal hygiene as in covering the mouth when coughing and sneezing, and disposal of nose and mouth secretions.
 - B. **Control of patient, contacts, and the immediate environment:**
 1. Report to local health authority: Official report not ordinarily justifiable, Class 5 (p. 6).
 2. Isolation: On first recognition of a common cold the infected person should avoid direct and indirect exposure of others, particularly little children, feeble or aged persons, or persons suffering from other illness. Such modified isolation as can be accomplished by rest in bed during the acute stage is advised.
 3. Concurrent disinfection: Of eating and drinking utensils; disposal of nose and mouth discharges, preferably by collecting on soft paper and burning.
 4. Terminal disinfection: Airing and sunning of room and bedding.

5. Quarantine: None.

6. Immunization of contacts: None.

7. Investigation of contacts and source of infection: Unprofitable.

8. Specific treatment: None. Indiscriminate use of antibiotics for uncomplicated colds or mild sore throat is to be discouraged. These valuable therapeutic agents should be reserved for complications such as pneumonia, tracheobronchitis, otitis, and sinusitis.

C. Epidemic measures: No effective measures for the control of epidemics are known. Isolation precautions may be helpful in institutions but procedures such as ultraviolet irradiation, aerosols, and dust control have not been effective.

D. International measures: None.

WHOOPIING COUGH

1. Identification—An acute bacterial infection involving trachea, bronchi and bronchioles, and characterized by a typical cough, usually of 1 to 2 months' duration. The initial catarrhal stage has an insidious onset with irritating cough which gradually becomes paroxysmal, usually within 1 to 2 weeks. Paroxysms are characterized by a repeated series of violent coughs, each series having many coughs without intervening inhalation and followed by characteristic crowing or high pitched inspiratory whoop; frequently ends with expulsion of clear, tenacious mucus. Young infants and adults often do not have the typical paroxysm. An absolute lymphocytosis is usual. The over-all fatality is low, less than 0.5%, but approximately 70% of deaths are among children under 1 year of age, with further concentration in those under 6 months, where fatality at times is as high as 30%. Synonym: Pertussis.

The infectious agent is readily recovered during catarrhal and early paroxysmal stages by nasopharyngeal swab.

Parapertussis is an allied disease clinically indistinguishable from whooping cough. It is usually milder and is relatively infrequent, incidence being indefinite. Identification is by immunologic differences between *Haemophilus parapertussis* and *H. pertussis*.

2. Occurrence—A frequent and common disease among children everywhere regardless of race, climate, or geographic location. In large communities incidence is generally highest in late winter and early spring; in smaller communities the seasonal incidence is variable. A marked decline in morbidity and mortality has occurred during the past 2 decades, primarily in those communities fostering extensive programs of active immunization and where good medical care is available.

3. Infectious agent—*Haemophilus pertussis*, pertussis bacillus.

4. Reservoir and source of infection—Man is the reservoir. Source of infection is discharges from laryngeal and bronchial mucous membranes of infected persons.

5. Mode of transmission—By direct contact with an infected person, by droplet spread, or indirectly by contact with articles freshly soiled with discharges of such persons.

6. Incubation period—Commonly 7 days, almost uniformly within 10 days, and not exceeding 21 days.

7. Period of communicability—Particularly communicable in early catarrhal stage before paroxysmal cough confirms provisional clinical diagnosis. After paroxysms are established, communicability gradually decreases and becomes negligible for ordinary nonfamilial contacts in about 3 weeks even though spasmodic cough with whoop may persist. For control purposes, the communicable stage is considered to extend from 7 days after exposure to 3 weeks after onset of typical paroxysms.

8. Susceptibility and resistance—Susceptibility is general; no good evidence of temporary passive immunity in young infants born of immune mothers. Whooping cough is predominantly a childhood disease, the incidence being highest under 7 years of age and mortality highest in infants, particularly those under 6 months. One attack confers a definite and prolonged immunity but second attacks occasionally occur, particularly in exposed adults. Fatality is higher in females than males at all ages. Both active and passive immunity may be induced by appropriate means.

9. Methods of control—

A. Preventive measures:

1. General immunization of all susceptible preschool children is an effective procedure for control of pertussis. Plain (preferred in Great Britain) or alum adjuvant vaccines may be used either alone or in combination with diphtheria and tetanus toxoids. Three doses of an alum adjuvant vaccine mixed with diphtheria and generally with tetanus toxoid, administered intramuscularly at 4-week intervals, beginning ideally at 1 to 2 months of age, is commonly used in the United States for simultaneous immunization against all 3 diseases. In general, routine immunization can be started at 3 to 4 months of age. The need for subsequent reinforcing doses of whooping cough vaccine is not definitely established, but when primary immunization is properly carried out in infancy, a single reinforcing dose is advised at 1 to 2 and again at 4 to 5 years of age; and additionally under circumstances of known direct exposure through familial contact. Infants living in institutions and in households with other susceptible children, particularly during the months when whooping cough is prevalent in the community, should have active immunization started by the time they are 2 months old.
2. Educational measures to inform the public and particularly parents of infants of the dangers of whooping cough and of the advantages of immunization in infancy.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report obligatory in most states and countries, Class 2B (p. 5).
2. Isolation: Separation of the patient from susceptible children and exclusion of the patient from school and public places for the recognized period of communicability. Isolation of children over 2 years of age is often impracticable; even for those under 2, should not be insisted upon at the expense of fresh air in the open if weather permits.
3. Concurrent disinfection: Discharges from the nose and throat and articles soiled therewith.
4. Terminal disinfection: Thorough cleaning.
5. Quarantine: Limited to exclusion of nonimmune children from school and public gatherings for 14 days after last exposure to a household or similar case; may be omitted if exposed nonimmune chil-

dren are seen by a physician or nurse on arrival at school each day for 14 days after last exposure. Particularly important to protect children less than 3 years old against contact with known or suspected whooping cough.

6. Immunization of contacts: Brief and limited passive immunity may be conveyed to young children by administration of appropriate amounts of hyperimmune gamma globulin or rabbit serum. Vaccination after effective exposure is of no proved value.
7. Investigation of contacts and source of infection: Carriers in the exact sense of the term are not known; search for missed and atypical cases among contacts is indicated.
8. Specific treatment: None; tetracycline antibiotics and chloramphenicol tend to shorten the infection, but not the symptoms, although minor amelioration may follow. These drugs are important therapeutically against secondary invaders, particularly those causing pneumonia.

C. Epidemic measures: A search for unrecognized and unreported cases is of value to protect preschool children from exposure and to assure adequate medical care for those exposed, especially infants. The comparatively high mortality among young infants justifies intensive effort toward their protection.

D. International measures:

1. Active immunization of susceptible infants and young children traveling to other countries, if not already protected; attention to desirable reinforcing dose for those previously inoculated.

YAWS

1. **Identification**—An acute and chronic relapsing nonvenereal treponematosi characterized by hypertrophic, granulomatous, or ulcerative destructive lesions of the skin, and by destructive and hypertrophic changes in bone. Three to 6 weeks after exposure an initial papule appears at the site of inoculation, eventually developing into a papilloma ("mother yaw"). In several weeks to months, and often before the initial lesion has healed, mild constitutional symptoms appear; also a generalized eruption of papules, some developing into typical frambesial lesions, in successive crops and lasting several months. Frambesial papillomata may appear on palms and soles, also hyperkeratoses in both early and late stages. The late stage often develops some years after the last early lesions with characteristic destructive lesions of skin and bone. Early and late lesions tend to heal spontaneously, but may relapse. Between these active phases the infection is latent, to give a latent early stage from which early relapses occur, and a latent late stage with late lesions and relapses. Central nervous system, eyes and viscera are not known to be involved, nor does congenital yaws occur. Rarely, if ever, fatal. Synonyms: Frambesia Tropica, Pian, Bouba, Parangi, and many others.

Diagnosis may be confirmed by dark-field examination of exudates from lesions. Serologic tests for syphilis are reactive with the same frequency in yaws as in syphilis, becoming positive during the initial stage, remaining positive during the early stage and tending toward non-reactivity after many years of latency, even without specific therapy.

2. **Occurrence**—Primarily a disease of rural people of tropics and subtropics; the lowest social and economic groups have the highest rates. Predomi-

nantly a childhood disease but occurs in later life; males outnumber females. Particularly common in Equatorial Africa, the Philippines, Burma, Vietnam, Laos, Cambodia, Indonesia, Thailand, and throughout the South Pacific Islands; endemic foci in the Caribbean area, in parts of Brazil, Colombia, Venezuela, Peru, Ecuador, Panama, and British Guiana; incidence is decreasing in many areas.

3. Infectious agent—*Treponema pertenue*.

4. Reservoir and source of infection—Man is the only reservoir. Source of infection is exudates of early skin lesions of infected persons.

5. Mode of transmission—Principally by direct contact. Indirect transmission by contaminated articles and flies is uncertain and probably of minor importance. *Transmission in utero is not established.*

6. Incubation period—From 2 weeks to 3 months; generally 3 to 6 weeks.

7. Period of communicability—Variable; may extend intermittently over several years while relapsing moist lesions are present; treponemata are not usually found in late ulcerative lesions.

8. Susceptibility and resistance—No evidence of natural or racial resistance. Infection results in immunity to homologous and heterologous strains, of slower evolution for heterologous strains and probably not complete until 1 year. The part of superinfection in nature is not well defined, and may be unimportant.

9. Methods of control—Although venereal syphilis has declined significantly in many areas, the nonvenereal treponematoses are a continuing problem. The infectious agents in all these disorders are morphologically and biologically almost identical and the resulting clinical syndromes come from epidemiologic rather than biologic differences.

A. Preventive measures: The following are applicable to yaws and to other nonvenereal treponematoses.

1. General health promotional measures; health education about treponematoses, better sanitation and improved social and economic conditions over a period of years will reduce incidence.
2. Organization of intensive control activities on a community basis, to include analysis of the specific local problem, clinical examination of entire populations, and mass treatment of patients with active lesions, latent cases and contacts. Periodic surveys and continuous surveillance are essential to successful results.
3. Provision of facilities for early diagnosis and treatment on a continuing plan, whereby the mass control campaign (9A2 above) is eventually consolidated into permanent local health services providing early diagnosis and treatment to patients, and contact investigation and health education to the community.
4. Emphasis on control of infectivity should not preclude treatment of disfiguring and incapacitating late manifestations nor the discovery and treatment of latent cases, since many late and latent infections subsequently relapse with infectious lesions to maintain the disease in the community.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: In selected endemic areas; in many countries not a reportable disease. Class 3B (pp. 5, 6). Differentiation of venereal and nonvenereal treponematoses with proper reporting of each has particular importance in evaluation of mass campaigns and in the consolidation period thereafter.
2. Isolation: None; avoid intimate personal contact until lesions are healed.

3. Concurrent disinfection: Care in disposal of discharges and articles contaminated therewith.
 4. Terminal disinfection: None.
 5. Quarantine: None.
 6. Immunization of contacts: Not applicable; prompt institution of a course of treatment (see 9B7 below).
 7. Investigation of contacts and source of infection: All familial contacts should be treated. Persons with no active disease should be regarded as latent cases and contacts, and treated. In areas of low prevalence, treat all active cases, all children, and close contacts of infectious cases.
 8. Specific treatment: Penicillin: For patients with active disease a single intramuscular injection of procaine penicillin G in oil with 2% aluminum monostearate (PAM) or benzathine penicillin G; half doses for children under 15 and for latent cases and contacts.
- C. Epidemic measures:** Active mass treatment programs are under way in many areas of high prevalence throughout the world. Essential features are (1) that a high percentage of the population be examined through field survey; (2) that treatment of active cases be extended to other segments of the population according to the demonstrated prevalence of active yaws; and (3) that periodic surveys be made at yearly intervals for 1 to 3 years, with surveillance and integration of activities into the established rural public health activities of the country.
- D. International measures:** To protect countries against risk of reinfection where active mass treatment programs are in progress, adjacent countries in the endemic area should institute suitable measures against yaws. Movement of infected persons across frontiers may need supervision.

YELLOW FEVER

1. **Identification**—An acute infectious disease of short duration and varying severity. The mildest cases are clinically indeterminate; typical attacks are characterized by sudden onset, fever, headache, backache, prostration, nausea and vomiting. As the disease progresses, the pulse rate slows in relation to temperature and albuminuria becomes pronounced; severe oliguria is frequent. Leucopenia appears early, most pronounced about the fifth day. Common hemorrhagic symptoms include epistaxis, buccal bleeding, hematemesis, and melena. Jaundice is moderate but post-mortem icterus may be pronounced. The fatality among indigenous populations of endemic regions is less than 5%; for persons of other origin, rates of 30 to 40% are common. One of the group of Arthropod-borne Viral Hemorrhagic Fevers, p. 34.

Laboratory procedures in diagnosis are isolation of virus from blood through animal inoculation; demonstration of a rising titer of neutralizing antibodies in paired acute phase and convalescent serums by test in mice; and demonstration of typical histopathological lesions of the liver.

2. **Occurrence**—Yellow fever transmitted by *A. aegypti* tends in the Americas to attack both sexes and all ages and races when first introduced into a community; in endemic urban yellow fever areas the disease is primarily one of childhood and of recently arrived foreigners. Jungle yellow fever of tropical America is predominantly a disease of adult males,

with the 20- to 40-year-age group most affected. Seasonal incidence follows the prevalence of local vector mosquitoes; commonest in rainy season. Geographical distribution of urban yellow fever has changed greatly since 1932 as a result of *Aedes aegypti* control campaigns. With the exception of a few cases in Trinidad, W. I., in 1954 and again in 1959, no aegypti-transmitted outbreaks have occurred in the Americas since 1942. Outbreaks of urban yellow fever are still reported from Africa. Available evidence indicates that jungle yellow fever is present from time to time in all countries of the mainland of the Americas, from Mexico south to Central and South America, with the exception of Uruguay and Chile where there are no tropical or subtropical forests with monkeys or other susceptible animals. In Africa it extends from the west coast south of the Sahara Desert through the Belgian Congo into Northern Rhodesia, Bechuanaland, Nyasaland, Uganda, Tanganyika, Kenya, Ethiopia, Somaliand, and Sudan. There is no evidence that yellow fever ever has been present in the Orient.

3. **Infectious agent**—The virus of yellow fever.

4. **Reservoir and source of infection**—In urban areas the reservoir of infection is man; in forest areas, vertebrates other than man, mainly monkeys, marmosets, possibly marsupials. Man has no essential part in transmission of jungle yellow fever, nor in maintaining the virus. The immediate source of infection for man is an infective mosquito.

5. **Mode of transmission**—In urban and certain rural areas, by the bite of the mosquito, *Aedes aegypti*. In forests of South America, by the bite of several species of forest mosquitoes of the genus *Huemagogus*, and *Aedes leucoclaenus*. In tropical Africa, *Aedes africanus* is the vector in the monkey population, while *Aedes simpsoni*, a semidomestic mosquito, and probably other *Aedes* species transmit the virus from monkey to man.

6. **Incubation period**—Three to 6 days.

7. **Period of communicability**—Shortly prior to onset of fever and for the first 3 days of illness the blood of patients is infective for mosquitoes. Highly communicable where many susceptible persons and abundant vector mosquitoes co-exist. Not communicable by contact or common vehicles. The extrinsic period of incubation in *A. aegypti* varies inversely with temperature, commonly 9 to 12 days at the usual summer temperatures. *A. aegypti* mosquitoes once infected remain so for life, but do not pass the virus to their progeny.

8. **Susceptibility and resistance**—Recovery from yellow fever is followed by lasting immunity; second attacks are unknown. Mild, inapparent infections are common in endemic areas. Transient passive immunity in infants born to immune mothers may persist up to 6 months. In natural infection, antibodies appear in the blood within the first week. Active immunity is induced by a suitable vaccine, see 9A3.

9. **Methods of control**—

A. Preventive measures:

1. Urban yellow fever can be completely prevented by eradication of *A. aegypti* mosquitoes. In the New World this is the indicated procedure, and has been accomplished in practically all of Central and South America. In North America and the Caribbean much still remains to be done.
2. Sylvan or jungle yellow fever, transmitted by forest species of *Aedes* and *Huemagogus*, cannot be controlled by any known method except vaccination, which is recommended for all persons living in

rural areas whose daily occupation brings them into forests in yellow fever areas, or for persons who intend to visit those areas.

3. Active immunization of all persons necessarily exposed to infection because of residence, occupation, or travel is necessary. Two methods of administering living modified vaccines are widely employed. A single subcutaneous injection of a vaccine prepared from the 17D strain of virus cultivated in chick embryo is effective. Antibodies appear from 7 to 10 days after vaccination and persist for at least 12 years, probably much longer. This vaccine is used exclusively in the Americas. Recent studies indicate that the 17D virus vaccine can be applied by scarification. A second method employs a vaccine combining living neurotropic yellow fever virus (Dakar strain prepared in mouse brain) with smallpox vaccine, administered by cutaneous scarification; used extensively and successfully in French West African territories, but reactions are more frequent and encephalitis is an occasional complication.

B. Control of patient, contacts, and the immediate environment:

1. Report to local health authority: Case report universally required by international regulation, Class 1 (p. 5).
2. Isolation: None; prevent access of mosquitoes to patient during first 3 days by screened sickroom or by spraying quarters with insecticide having residual effect, or by bed net.
3. Concurrent disinfection: None; home of patient and all houses in vicinity should be sprayed promptly with a residual insecticide such as DDT, benzene hexachloride, chlordane, or dieldrin.
4. Terminal disinfection: None.
5. Quarantine: None.
6. Immunization of contacts: Family and other contacts and neighbors not previously immunized should be vaccinated promptly.
7. Investigation of contacts and source of infection: Inquiry about all places, including forest areas, visited by patient 3 to 6 days before onset, to locate focus of yellow fever; observation of all persons visiting that focus. Search of premises and place of work for mosquitoes capable of transmitting infection. Attention to mild febrile illnesses and unexplained deaths suggesting yellow fever.
8. Specific treatment: None:

C. Epidemic measures:

1. Urban or *Aedes aegypti*-transmitted yellow fever:
 - a. Mass vaccination beginning with most exposed persons and those living in *aegypti*-infested parts of the area.
 - b. Rapid spraying of all houses in community with residual insecticide such as DDT, benzene hexachloride, chlordane or dieldrin.
 - c. Application of larvicide to all actual and potential breeding places of *A. aegypti*.
2. Jungle or sylvan yellow fever:
 - a. Immediate vaccination of all persons living in or near forested areas or entering such areas.
 - b. Avoidance by unvaccinated individuals of those tracts of forest where infection has been localized, and similarly by vaccinated persons for the first week after vaccination.

3. In regions where yellow fever may occur, a viscerotome service should be organized to collect for diagnostic purposes small specimens of liver from fatal febrile illnesses of 10 days duration or less: many cases and outbreaks otherwise missed are thereby discovered.
4. Deaths of howler and spider monkeys in the forest, when confirmed, are presumptive evidence of the presence of yellow fever. Confirmation by histopathological examination of livers of moribund or recently dead monkeys is highly desirable.
5. Immunity surveys by mouse neutralization test of wild primates captured in forested areas are useful in defining enzootic areas. Serological surveys of human populations are practically useless nowadays because of the wide use of yellow fever vaccination.

D. International measures:

1. Telegraphic notification to WHO and to adjacent countries by governments of the first case of yellow fever arising within the country and of newly discovered or reactivated foci of yellow fever infection among vertebrates other than man.
2. Measures applicable to ships, aircraft, and land transport arriving from yellow fever areas are specified in International Sanitary Regulations, World Health Organization, annotated edition, Geneva, 1957.
3. Animal quarantine: Quarantine of monkeys, marmosets and other wild primates arriving from yellow fever areas may be required until 7 days have elapsed after leaving such areas.
4. International travelers: A valid antiyellow fever vaccination certificate is required by many countries for entry of travelers coming from or through recognized yellow fever zones of Africa and South America; otherwise, quarantine measures are applicable. The international certificate of vaccination is valid from 10 days after date of vaccination and for 6 years; if revaccinated within that time, from date of that revaccination and for 6 years.

INDEX

A		<i>Page</i>		<i>Page</i>
Abscess ...	21, 23, 78, 82, 120,	175	Bat, unspecified	202
Actinomycosis		19	Fruit eating	143
Acute respiratory disease (ARD),			Insectivorous	143
of military recruits.....		216	Vampire	143
Adenovirus infection	215,	216	Bauru ulcer	100
African sleeping sickness		199	Bear	196
African tick-borne fever, rick-			Polar	196
ettsial		20	Bejel	185
African tick typhus		20	Benzene hexachloride	111,
Air-borne transmission	25,			160, 202,
46, 57, 58, 61, 82, 93, 95, 114,				225
120, 133, 140, 142, 153, 161,			Bilharziasis	165
170, 172, 174, 178, 204, 211,		213	Birds, unclassified	30, 32,
Alastrim		169		34, 35, 140,
Aldrin		152	Water	106
Aleppo boil		100	Blastomycosis	
Amebiasis		21	European	61
Amebic dysentery		21	North American	42
Ancylostomiasis		23	South American	43
Animal, unclassified..	19, 25, 32,		Blennorrhoea, inclusion	87
44, 47, 49, 55, 58, 61, 62, 69,			Blindness	87, 121, 194
93, 122, 126, 142, 155, 156,			Boil	173, 175
163, 191, 193, 196, 199, 202,		207	Bone meal	26, 27, 163
Antelope		199	Bornholm disease	131
Anthrax		25	Boston exanthem	161
Armadillo		202	Botulism	44, 80
Arthritis, gonococcal		84	Bouba	221
Arthropod-borne viral hemor-			Boutonneuse fever	20
rhagic fevers	34,	223	Breakbone fever	62
Arthropod-borne viral encephal-			Brill's disease	212
itides		30	Brucellosis	45
Arthropod-borne viral fevers ...			Bug, cone-nosed or triatomid...	202
33, 58, 62,		153	Bull snake	207
Arthropod-borne viral infections,			Bunyamwera fever	33
A summary		27	Burro	57
Arthropod, unclassified, transmis-			Bushbuck	199
sion by	108, 127, 153,	193	Bwamba fever	33
Articular rheumatism, acute		150		
Ascariasis		38		
Aseptic meningitis syndrome...		39		
Athlete's foot	154,	157		
Azul		127		
B			C	
Baghdad boil		100	Calabar swelling	107
Balantidiasis		21	California encephalitis	29
Bandicoot		142	Camel	74
Bartonellosis		41	Candidiasis	47
			Cane field fever	105
			Canicola fever	105
			Caraparu fever	29
			Carate	127
			Carbuncle	173
			Caribou	74

	Page		Page
Carrier	41, 48, 50, 52, 60, 67, 72, 85, 89, 91, 106, 116, 124, 133, 134, 140, 163, 164, 174, 176, 177, 178, 181, 200, 209, 215, 221	Copepod	69
definition	11	Coryza	214, 218
Cat	49, 61, 93, 98, 100, 102, 122, 140, 143, 155, 193, 202, 207	Coxsackie virus infection....	40, 62, 131
Cat scratch fever	48	Coyote	143, 207
Catarrhal conjunctivitis	215	Crab	122
Catarrhal fever	216	Crayfish	122
Cattle	19, 25, 46, 47, 57, 61, 74, 106, 140, 142, 153, 155, 166, 189, 193, 199, 204	Cream, synthetic	124
Catu fever	29	Creeping eruption	24
Cerebrospinal fever	116	Crimean hemorrhagic fever.....	35
Cervicitis, gonococcal	84	Crustacea	70
Chagas disease	201	Cryptococcosis	61
Chancre	186	Cyclops	70
Chancre mou	50	Cysticercosis	189
Chancroid	50		
Charbon	25	D	
Chemoprophylaxis	73, 87, 112, 113, 117, 130, 133, 135, 151, 169, 179, 200, 206	DDT	42, 63, 79, 111, 121, 125, 130, 147, 160, 202, 212, 213, 225
Chicken	93, 163, 193	Deer	207
Chickenpox	51	Definitions	11
Chiclero ulcer	100	Delhi boil	100
Chikungunya fever	33	Dengue	34, 62
Chinese liver fluke disease.....	55	Dermatitis verrucosa	54
Chlordane	160, 168, 225	Dermatomycoses, the	154
Cholera	52	Dermatophytoses, the	154
Choriomeningitis, lymphocytic..	108	Devil's gripe	131
Chromoblastomycosis	54	Diarrhea, of the newborn, ep- idemic	63
Chromomycosis	54	Dichuchwa	188
Chronic venereal sore.....	88	Dieldrin....	111, 160, 168, 202, 225
Cleaning, definition	11	Diethyltoluamide	21, 112, 160, 168
Climatic bubo	109	Dimethylphthalate	21, 107, 112, 149, 160
Clonorchiasis	55	Dingo	74
Coccidioidomycosis	56	Diphasic meningoencephalitis ..	32
Cockroach	163	Diphasic milk fever	32
Colitis, ulcerative	21	Diphtheria	66
Colorado tick fever, viral....	34, 58	Diphyllbothriasis	69
Common cold	218	Disinfection, definition	12
Communicable disease, definition	11	Disinfestation, definition	12
Communicable period, definition	11	Distomiasis, pulmonary	122
Conjunctivitis		Dog	20, 42, 55, 57, 61, 69, 74, 77, 93, 98, 100, 102, 106, 108, 122, 143, 155, 159, 166, 184, 185, 193, 202, 207
Acute bacterial	59	Donkey	81
Acute of the newborn.....	87	Donovaniasis	88
Catarrhal	215	Dracontiasis	70
Chronic follicular	194	Duck	140, 163
Contact, definition	11	Dust, transmission by....	26, 39, 42, 43, 57, 58, 61, 64, 75, 93, 108, 120, 142, 172, 178, 191, 204, 211, 213
Contamination, definition	12	definition	17
Control measures, definition....	3		

	Page		Page
Dysentery		Mango fly	107
Amebic	21	Sandfly	41, 100, 102, 126
Bacillary	71	Tsetse fly	199
E		Food handlers	53, 68, 73, 81, 163, 179, 190, 210
Eastern equine encephalitis	30	Food poisoning	80
Echinococciasis	73	Botulism	44
Echinococcosis	73	Salmonella	162
Echo virus infection	40, 161	Staphylococcal	80
Education in personal cleanliness, definition	12	Welch bacillus	80
Elephantiasis	78	Food, transmission by	22, 26, 39, 44, 52, 55, 69, 72, 75, 77, 80, 89, 92, 93, 108, 123, 124, 137, 163, 178, 189, 196, 208, 209
Empeines	127	definition	16
Encephalitis, arthropod-borne		Foot and mouth disease	82
viral	30	Fort Bragg fever	105
Mosquito-borne	30	Fox	93, 106, 143, 196, 202, 207
Tick-borne	32	Frambesia tropica	221
Encephalitis, other forms	137	Fumigation, definition	13
Encephalitis, herpetic	30	G	
Encephalitis lethargica	30	Gammexane	202
Encephalitis, postvaccinal	30, 144, 225	Gastroenteritis	80
Encephalitis, postinfection	30, 51	Gerbil	100, 102
Enteric fever	209	German measles	160
Enterobiasis	75	Germiston fever	29
Epidemic, definition	12	Giardiasis	21
Epididymitis	78	Glanders	81
Gonococcal	84	Glandular fever	117
Erysipelas	177, 180	Gnat, eye	60
Erysipeloid	181	Goat	25, 46, 140, 142, 153, 193
Erythema infectiosum	160	Gonococcal infection	83
Espundia	100	Gonorrhea	84
Exanthem subitum	161	Gonorrheal ophthalmia	86
F		Granuloma	
Farcy	81	Coccidioid	57
Fascioliasis	77	Contagiosa	88
Fasciolopsiasis	76	Inguinale	88
Fatality, definition	12	Paracoccidioid	43
Favus	154	Pudenti tropicum	88
Febrile catarrh	216	Ulcerating	88
Ferret	202	Venereum	88
Fifth disease	160	Ground itch	24
Filariasis	78	Guama fever	29
Finch	140	Guinea pig	108
Fish	55, 69, 181	Guinea worm disease	70
Fish or broad tapeworm infection	69	H	
Fleas	128, 211	Hair	25, 155
Flies, transmission by domestic	22, 52, 60, 72, 92, 100, 124, 132, 194, 209, 222	Hare	35, 207
Biting	25	Haverhill fever	145
Black fly	121	Hemoptysis, endemic	122
Deer fly	207		

Page

Hemorrhagic fever, epidemic...	37
Hemorrhagic nephroso-nephritis.	37
Hepatitis	
Epidemic	89
Infectious	89
Serum	90
Herpangina	91
Herpes encephalitis	30
Herpes simplex	40
Herpes zoster	51
Heterophydisias	55
Hides	25
Histoplasmin	93
Histoplasmosis	92
Hookworm disease	23
Horse.....	19, 25, 30, 42, 46, 57, 61, 81, 155, 156, 191
Host, definition	13
Hydatidosis	73
Hydrophobia	143

I

Ilesha fever	29
Ilheus fever	28
Ilheus encephalitis	28
Immune person, definition.....	13
Immunization	
Active.....	26, 31, 33, 37, 53, 67, 95, 101, 106, 119, 124, 129, 133, 138, 142, 144, 160, 168, 170, 179, 191, 205, 208, 210, 212, 213, 216, 217, 220, 224, 225
Passive	
Animal immune serum....	31, 144, 221
Antitoxin	45, 68, 179, 191
Convalescent serum	31
Gamma globulin	114, 139, 162, 217, 221
Immune serum globulin...	90, 114, 138, 162
Impetigo	
Contagiosa	173
Of the newborn.....	173
Inapparent infection, definition..	13
Incidence, definition	13
Incubation period, definition...	13
Indalone	139
Infantile enteritis	63
Infantile paralysis	137
Infected person, definition.....	13
Infection, definition	13
Infectious agent, definition.....	13
Infectious disease, definition....	13

Page

Infestation, definition	13
Influenza	94
Insect, unspecified	137
Insecticide, unspecified	121, 126, 160
Aerosol spraying	101, 111
Definition	13
Liquid spraying	112
Residual action	31, 42, 63, 79, 101, 103, 111, 112, 125, 130, 147, 152, 168, 200, 202, 212, 213, 225
Isolation, definition	14

J

Jackal	100, 102
Japanese B encephalitis	30
Jaundice	
Catarrhal	89
Epidemic	89
Hemorrhagic	105
Homologous serum	90
Jockey itch	154

K

Kala azar	102
Kenya dog fever.....	20
Keratitis, infectious punctate....	97
Keratoconjunctivitis, epidemic...	97
Kyasanur forest disease.....	36

L

Larva migrans visceral.....	98
Latent infection, definition	13
Larvicide	112
Leishmaniasis	
Cutaneous	100
Visceral	102
Lepromin	104
Leprosy	103
Leptospirosis	105
Lice	124, 147, 213
Lindane	125, 160, 213
Loaiasis	107
Lota	127
Louping ill	32
Lousiness	124
Lung fluke disease, Oriental	122
Lymphocytic choriomeningitis...	30, 108
Lymphogranuloma inguinale	109
Lymphopathia venereum	100
Lymphoreticulosis, benign	40

M	Page
Madura foot	120
Maduramycosis	120
Malaria	110
Mal del pinto	127
Malignant pustule	25
Mallein	82
Malta fever	46
Mammal, marine	196
Marituba fever	29
Marmoset	224
Marsupial	224
Mayaro fever	33
Measles	113
Mediterranean fever	46
Melioidosis	82
Meningismus	116
Meningitis	
Candidial	47
Chronic	40
Coccidioidal	57
Cryptococcal	61
Leptospiral	105
Meningococcal	115
Nocardial	120
Nonbacterial	40
Purulent secondary	116, 173, 175
Salmonella	162
Serous	40
Syphilitic	137
Torula	61
Tuberculous	203
Viral	40
Meningitis syndrome, aseptic	39
Meningococemia	115
Meningoencephalitis	33, 117
Diphasic	32
Mice :-	108, 151, 152, 159, 166, 193
Middleburg disease	153
Midge	126
Milk, or milk products, trans-	
mission by	32,
46, 53, 67, 72, 80, 89, 124,	
137, 142, 146, 163, 178,	
204, 209	
definition	16
Mite	27, 37, 152, 164, 168
Molluscicide, definition	14
Monihiasis	47
Mongoose	37, 61,
Mongoose	108, 111, 202, 224
Monoeytic angina	117
Mono-nucleosis, infectious	117
Moose	74
Morbidity, definition	14
Morbilli	114

	<i>Page</i>
Mortality, definition	14
Mosquito 27, 30, 34, 62, 78, 111, 132, 153, 208,	224
Mud fever	105
Mule	81
Mumps 40,	118
Murine typhus	211
Murray valley encephalitis	30
Muskrat 37,	207
Myalgia, epidemic	131
Mycetoma	120

N

Nairobi sheep disease.....	28
Nephroso-nephritis, hemorrhagic.	37
Njovera	188
Nocardiosis	120

O

Omsk hemorrhagic fever.....	36
Onchocerciasis	120
Ophthalmia neonatorum	
Gonococcal	86
Nonspecific	86
Opisthorchiasis	55
Opossum	93, 106, 202, 207
Oriboca fever	29
Oriental lung fluke disease....	122
Oriental sore	100
Ornithosis	140
Oroya fever	41
Oxyuriasis	75

P

Pappataci fever	126
Paragonimiasis	122
Parakeet	140
Parangi	221
Parapertussis	219
Paratyphoid fever	123
Parotitis, infectious	119
Parrot	140
Pathogenicity, definition	14
Patient or sick person, definition	14
Pediculosis	124
Pemphigus neonatorum	173
Peritonitis, pelvic gonococcal	84
Pertussis	219
Pestis minor	129
Petrel	140

Page

Page

Pharyngitis		Raccoon	106, 143, 202
Acute febrile	215	Radesyke	188
Nonbacterial	215	Rat	82, 93, 106, 129, 146, 166, 196, 202, 207, 211
Nonbacterial exudative (and tonsillitis)	215	Rat-bite fever	145
Streptococcal, hemolytic	177	Reindeer	74
Pharyngoconjunctival fever	215	Relapsing fever	
Philippine hemorrhagic fever	34	Louse-borne	147
Phlebotomus	27, 41, 100, 102, 126	Tick-borne	148
Phlebotomus fever	34, 126	Repellent, insect	21, 63, 79, 101, 103, 107, 112, 121, 127, 131, 160, 168
Pian	221	definition	15
Piedra	154	Report of a disease, definition	15
Pigeon	61, 93, 140	Reporting of communicable disease	4
Pink eye	59	Case report	5
Pinta	127	Collective report	5
Pinworm disease	75	Epidemic report	5
Plague	128	Reservoir of infection, definition	15
Plant, reservoir of infection	43, 54, 57, 172	Resistance, definition	15
Pleurodynia	131	Respiratory disease	
Pneumonia	94	Acute febrile of unknown origin	217
Acute lobar	132	Acute of military recruits, (ARD)	216
Bacterial	134	Acute upper	214
Bronchial	132	Rheumatic fever	150, 179
Pneumococcal	132	Rice field fever	105
Primary atypical	135	Rickettsialpox	151
Virus	135	Rift valley fever	34, 152
Pneumonitis	61, 82, 98	Ringworm	154
Poliomyelitis	137	Rocky mountain spotted fever	159
Porcupine	58	Rodent, unspecified	20, 32, 37, 106, 128, 146, 149, 153, 155, 159, 163, 166, 168, 193, 202
Poultry unclassified	140, 163	Rodenticide, definition	16
Powassan encephalitis	28	Roseola infantum	161
Preface	1	Rubella	160
Prevalence, definition	14	Rubeola	114
Proctitis, nongonococcal	85	Russian spring-summer encephalitis	32
Prostatitis, gonococcal	84	Rutgers "612," insect repellent	112, 149
Psittacosis	140		
Puerperal fever	177		
Streptococcal	182		
Puerperal septicemia	182		
Pulmonary distomiasis	122		
Pyrethrum	112		

Q

Q fever	141
Quail	207
Quarantil fever	29
Quarantine, definition	15

R

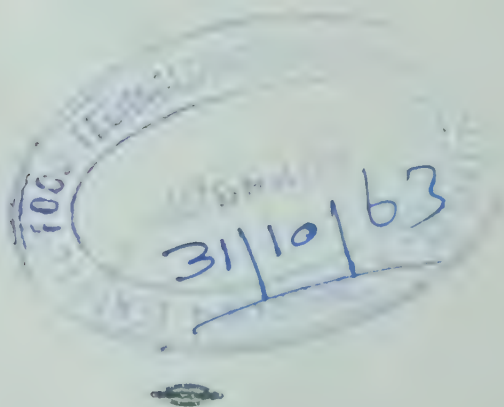
Rabbit	159, 207
Rabies	143

S

Sage hen	207
St. Louis encephalitis	30
Salmonellosis	162
Salpingitis, gonococcal	84
Sandfly, see Phlebotomus	

	Page		Page
Sandfly fever	126	Taeniasis	189
Scabies	164	Tapeworm	
Scarlet fever	177	Beef	189
Schick test	67	Diminuta	189
Schistosomiasis	165	Dipylidium caninum	189
Scrub typhus	167	Dog	74
Seatworm disease	75	Hymenolepis nana	189
Semliki forest fever	28	Pork	189
Sheep	25, 46, 57,	Tetanus	190
74, 77, 140, 142, 153, 193,	207	Neonatorum	191
Shellfish	124, 209	Thailand hemorrhagic fever, ...	34
Shigellosis	71	Three-day fever	126
Sibbens	188	Thrush	47
Sixth disease	161	Tick	20, 27, 32, 35,
Sunk	93, 106, 143, 207	36, 37, 58, 142, 149, 159, 193,	207
Sleeping sickness, African	199	Tick-bite fever, South African ..	20
Smallpox	169	Tina	127
Sore nail	55, 77, 122, 166	Tinea	154
Sodoku	146	Tonsillitis	
Soft chancre	50	Nonbacterial exudative (and	
Soft sore	50	pharyngitis)	215
Sol	24,	Streptococcal	177
25, 38, 42, 43, 44, 54, 57,		Torulosis	61
93, 99, 120, 172, 184, 190,		Toxoid reaction test	67
191, 198		Toxoplasmosis	192
Sore eyes	59	Trachoma	194
Sore throat, streptococcal	177	Transmission of infection	
Source of infection, definition ..	16	Air-borne	17
South African tick-bite fever ..	20	Contact	16
Spirillum minus infection	146	Definition	16
Spondweni fever	28	Vector	16
Sporotrichosis	172	Vehicle	16
Squirrel	58, 146, 207	Trichinellosis	195
Staphylococcal infection	173	Trichiniasis	195
General, often hospital-ac-		Trichinosis	195
quired	175	Trichocephaliasis	198
Stomatitis		Trichomoniasis	197
Gangrenous	102	Trichuriasis	198
Herpetic	92	Trypanosomiasis	
Streptobacillus moniliformis in-		African	199
fection	145	American	201
Streptococcal infection, hemolytic	177	Tsutsugamushi disease	167
Strongyloidiasis	184	Tuberculin	204
Subclinical infection	13	Tuberculosis	203
Susceptible, definition	16	Disseminated or extrapulmo-	
Aspect, definition	16	nary	203
Swimmer's itch	166	Meningeal	207
Swine	19, 25, 30, 46,	Miliary	207
55, 57, 74, 77, 106, 108, 122,		Pulmonary	203
166, 189, 193, 196, 199, 202,	207	Tularemia	207
Winchard disease	105	Turkey	140
Phylis	185	Typhoid fever	209
Nonvenereal	188	Typhus abdominalis	209

	Page		Page
Typhus exanthematicus	212	Vulvovaginitis	47
Typhus fever		Gonococcal of children.....	85
African tick	20	Nonspecific	85
Epidemic, flea-borne	211		
Epidemic or classical (louse- borne)	212	W	
Miteborne	167	Water buffalo	166
Murine typhus	211	Water, transmission by.....	22, 52, 70, 72, 74, 89, 106, 124, 137, 166, 208, 209
		definition	16
U		Waterhouse-Friderichsen syn- drome	115
Uganda S fever.....	29	Weasel	146
Ulcus molle	50	Weil's disease	105
Uncinariasis	23	Wesselsbron disease	29, 153
Undulant fever	46	West Nile fever.....	33
Upper respiratory disease	214	Western equine encephalitis ...	30
Urethritis gonococcal	84	Whipworm disease	198
Uruma fever	28	Whooping cough	219
Uta	100	Wolf	74, 143, 196
		Woodchuck	207
V		Wool	25, 142
Vaginitis			
Candidial	47	Y	
Trichomonal	197	Yaws	221
Valley fever	56	Yellow fever	35, 223
Varicella	51		
Variola	169	Z	
Venezuelan equine encephalitis	28	Zika fever	29
Venezuelan equine fever.....	33	Zoonosis, definition	17
Verruga peruana	41		
Vincent infection	66		
Vulture	25		



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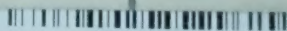
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